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RECENT TRENDS IN TUBERCULOSIS IN SOUTH AUSTRALIA.[1]

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PROGRESS in medicine, as in other branches of science, has characteristically followed the introduction and exploitation of new techniques. In the naming of this society we pay homage to one who introduced a new technique—auscultation by means of the stethoscope—and who, in exploiting this technique, not only described practically all the physical signs we employ in chest examination today, but also correlated these with the underlying pathological processes. Laennec died of tuberculosis at the age of 45 years, having laid the foundations of modern chest medicine.

Just 10 years later the first white settlers arrived in South Australia, and in a very short time the same disease which had caused the death of the young French physician made its presence felt in the new colony. After only three years spent in laying out this city and surveying the surrounding country, Colonel William Light died of tuberculosis in 1839. In 1842, when the Registrar-General began to keep records, consumption soon figured prominently among the causes of death. In fact the fourth recorded death in the first volume of registrations is that of a boy aged six years who died of consumption. As the young colony grew and urban conditions developed, tuberculosis took a heavier and heavier toll, especially of the younger people. The average age at death of the first 20 victims of tuberculosis recorded in the Adelaide register was 28·7 years. Since that time tuberculosis has continued to be a major cause of invalidism and mortality, but its impact has changed in a number of striking ways.

It is my purpose to examine the available evidences of these changes in recent years, and to attempt to draw some conclusions from the observations presented. I do not propose to discuss in detail the techniques by which information about tuberculosis has been gained. Until 65 years ago all our information on the subject came from the physician and the morbid anatomist. The contributions of bacteriology and radiology, and more recently of surgery, have clarified the natural history of the disease in the individual to a point where one is almost tempted to think at times that we are beginning to understand it. At the same time the epidemiologist, by summing up these individual experiences, seeks to present an intelligible picture of the impact of the disease on the community as a whole.

Mortality.

The first type of data which provides information for the epidemiologist relates to mortality. The crude death rate, that is, the number of people per 100,000 of population

[1] Presidential address to the South Australian Branch of the Australian Laennec Society, September 19, 1957.

dying each year from tuberculosis, is of some interest as it shows a progressive decrease during the whole of this century (Figure I). The change appeared to be going on before either the treatment of individual patients or public health measures designed to reduce the spread of infection were likely to have any effect at all. It is interesting to speculate to what extent general social betterment may have been responsible, or whether we were witnessing a natural change in balance between host and parasite. However, in the last 10 years, the trend of the curve does seem to have changed. Instead of flattening out, it appears to have become rather steeper. This change in trend has coincided with the introduction of chemotherapy and the surgical attack on the disease.

If we turn now to age-specific death rates, we see a much more informative picture. In a rapidly changing population such as ours, subjected to a constant trickle as well as sudden great waves of migration and to two major wars, a reasonably accurate picture of the age-distribution of the population is possible only in years when a census has been taken. We are therefore able to obtain accurate age-specific or group death rates only in census years. When we divide our small population into age and sex groups, even using ten-year age groups, we find ourselves dealing with small collections of people, and the number of deaths from tuberculosis in each group may be very small indeed.

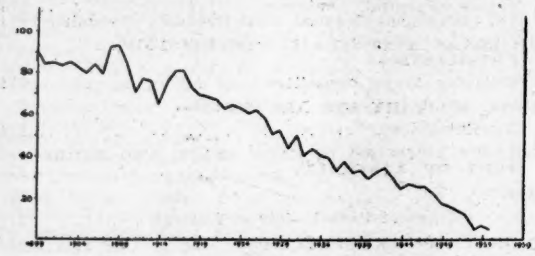

FIGURE I.

Number of deaths from tuberculosis each year per 100,000 of population.

Small chance variations in deaths from year to year may therefore make quite a big difference in death rates. In order to overcome these variations, the Government statistician has calculated age-specific death rates from tuberculosis for the last five census years, 1911, 1921, 1933, 1947 and 1954 using in each case the group population of the census year and the average number of deaths per year for three years—the census year and the years immediately preceding and succeeding.

Figures II, III and IV are constructed from these group death rate tables. Figure II shows group death rates from respiratory tuberculosis in males. It will be seen that each successive census year shows not only a decrease in mortality, as was indicated in our crude death rate graph, but also a progressive shift to the right, that is, to the older age groups. Tuberculosis is no longer a significant cause of death in young men, and it has moved almost entirely into the period beyond middle age, when its association with increasing fibrosis and emphysema is common.

I should also draw your attention to the fact that a diagonal line drawn from the younger age groups in 1911 through the middle ranges in 1921 and 1933 and into the older age groups in 1947 and 1954 serves to trace the mortality experience of the generation born towards the close of last century. If our diagram had embraced a longer period of time, we might have seen definitely what it begins to suggest, that is, that the apparent change in tuberculosis from a fatal disease of young and middle-aged men to a fatal disease of old men, may represent only the passing of a generation of people who have been susceptible to, or subjected to, this disease at every stage of their lives.

Turning now to group death rates from respiratory tuberculosis in females, we see in Figure III a clearer, less ambiguous and rather pleasanter picture. In the 1911 curve the high death rate in young women stands out very prominently, but this peak of mortality has not moved to the right and fatal tuberculosis has not followed that

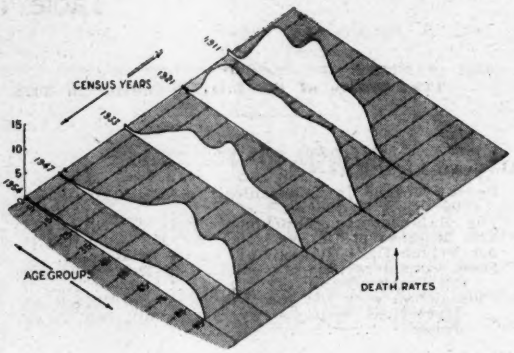

FIGURE II.

Group death rates from respiratory tuberculosis per 10,000 males in decennial age groups.

generation throughout its life span. Perhaps all the susceptibles were killed off in their early years. At all events, the reputation of tuberculosis as a killer of young women is no longer deserved, in fact the condition is virtually no longer lethal for the female at all.

Group mortality rates from non-respiratory tuberculosis are similar in the two sexes, so I have added them together

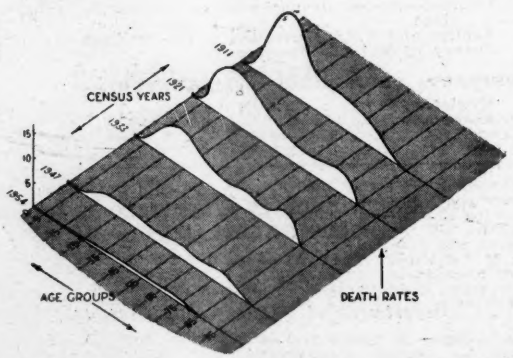

FIGURE III.

Group death rates from respiratory tuberculosis per 10,000 females in decennial age groups.

in one diagram (Figure IV). The most significant cause of death in these groups has been meningitis and miliary tuberculosis in young children. Even in the earlier curves there was a marked decline in mortality in the group aged under five years, undoubtedly due to the decreasing incidence of miliary disease. The effect of chemotherapy in revolutionizing the prognosis of these conditions is seen only in the most recent curve.

There is one other important aspect of mortality figures which I would like to touch upon briefly. In the early days of chemotherapy and surgery for tuberculosis, it was often suggested that the dramatic decrease in death rates represented merely a temporary postponement of an inevitable result. I suppose in a sense this is true; but if the postponement is such that most patients live a normal life span and die of some unrelated condition, then the view put forward is not a very realistic one.

One way to investigate this point is to see whether any change is occurring in the length of time between notification and death from tuberculosis.

In 1956 there were 32 deaths from pulmonary tuberculosis of patients whose condition had been notified prior to death. The average interval between notification and death of these patients was 6·6 years. For comparison I have chosen the first 32 deaths from tuberculosis in 1950, when the average interval was 4·8 years.

It therefore seems true that modern treatment is postponing death in those who do finally die of tuberculosis; but at the same time the figures I have shown earlier indicate such a great decrease in total mortality as to completely overshadow this small effect.

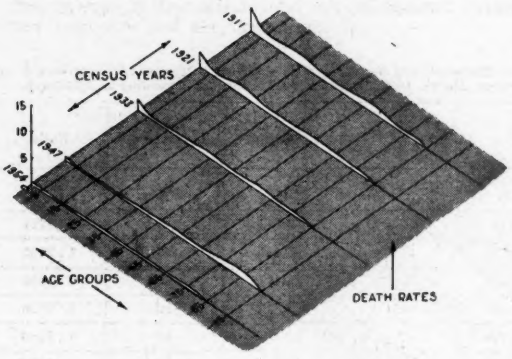

FIGURE IV.
Group death rates from non-respiratory tuberculosis for both sexes in decennial age groups.

Morbidity.

There is an air of finality about mortality figures which is very different from the information available about tuberculosis and tuberculous infection in the living community. Morbidity data are generally less definite, but perhaps more interesting because they represent live people and situations.

In a disease of brief duration, the prevalence, that is, the number of cases per unit of population at a given time, is likely to be much less than the annual incidence. But in a long-continuing condition like tuberculosis the reverse is true; prevalence greatly exceeds annual incidence. In either case these two yards-sticks of morbidity have to be considered separately.

The true prevalence of tuberculosis in the community is indicated by the number of sufferers in relation to the total population. But the actual number of sufferers at any one time is extremely difficult to determine. There is often a long interval between the onset of the disease and its discovery, and sometimes a much longer interval before the case is notified, though I believe there has been a very sharp drop in the time lag between diagnosis and notification in recent years. This reflects in part the increased interest in tuberculosis work, but perhaps even more the fact that the patient no longer feels he has much to lose and nothing to gain by being notified. Patients, and more particularly almoners, are well aware of the better scale of financial support for tuberculosis sufferers than for invalids of other types. One effect, then, of special tuberculosis allowances has been to make the notification of new cases of tuberculosis almost complete.

But in trying to determine the prevalence of tuberculosis, one of the greatest difficulties is to decide when a patient should no longer be regarded as suffering from the disease.

Health Act Regulations allow any medical practitioner to give a certificate of clearance to a person previously notified as suffering from tuberculosis. This certificate generally states that the patient is no longer suffering from active tuberculosis and is not in danger of infecting other people. I need hardly stress the difficulty of deciding when this point has been reached. Some patients are very concerned to obtain a certificate of clearance, not only because it gives them a feeling of confidence in the future, but also because it may have an important influence on their employment or on their relations with friends or neighbours or a landlord. But many others have no interest in such a document and take no steps to obtain one.

Our practice is to retain the record of each notified patient in our case registry until five years after his receipt of a clearance certificate or until the patient dies or moves away from South Australia.

Therefore, prevalence figures based on the case registry will be deficient on account of cases not yet diagnosed or not notified, and they will be excessive on account of patients whose disease has been arrested for many years, but who have not bothered to obtain a certificate of clearance. We can give no close estimate of the extent to which these two errors cancel each other out, but there are at present 3729 names in the central tuberculosis case registry. In a population of 862,000 this represents a prevalence of 433 per hundred thousand or one in every 231 persons.

But there is now another means by which we can arrive at an estimate of the prevalence of tuberculosis. Since March, 1952, we have been conducting compulsory X-ray surveys of the population aged 14 years and over. At first, those over 65 years were not required, but merely invited to attend. This was later raised to 70 years; and in the last two years there has been no upper age limit.

A very large proportion of those required to attend do so; for example, in a recent survey of that part of Port Adelaide east of the Port River, it was estimated that 12,000 persons would be required to attend. In fact, 12,133 persons were examined in the survey, and 115 others from the area attended the Adelaide City unit during the same period. However, there were 900 names on the electoral roll of persons who had apparently not attended. These were all questioned by letter, and the following results were obtained: 219 persons had recently been examined in an adjacent survey; 182 immediately took steps to be examined in the succeeding survey or privately; 195 were either in hospital or bedridden at home; 240 had left the area of the compulsory survey; 26 had been examined, but had given names differing slightly from those on the electoral roll; and 38 failed to reply; so 1·6% were too ill to attend and 0·32% could not be accounted for.

While it is possible that there was a high prevalence of tuberculosis among the 2% not examined, we can obtain valuable information about the prevalence of the disease from the findings in 98% of the population in this survey and a similar proportion over a much wider area of the State, over which the five-year period from March, 1952, to March, 1957. During this period almost 300,000 persons have been examined, considerably more than half of them in the metropolitan area.

In the country a good deal remains to be done in the mid-north and within 50 miles of Adelaide and in the Mallee district, but all the large towns and the remote areas of the State have been covered.

It will be seen in Table I that 2·8% of films are reported as showing abnormalities probably of tuberculous origin. This includes a large number of calcified primary complexes and many other lesions which are quite inactive. But the total figure is probably too low, as our radiologists sometimes report on films showing small calcified spots as "no abnormality requiring further investigation".

As a result of investigation after the finding of abnormalities in survey films, 262 new cases of tuberculosis have been notified. This represents a prevalence of previously unknown active tuberculosis of 0·089% or one in every 1124 persons examined. In addition, we see approximately four times as many films of tuberculous patients who had previously been notified; but this number

is not an accurate index of notified cases in the community examined, because some of these people submit evidence of recent X-ray examination instead of attending the surveys.

TABLE I.

"Total Population" X-ray Surveys in South Australia, March, 1952, to March, 1957.

Site of Survey.	Number of Persons Examined.	Abnormalities Probably of Tuberculous Origin.		New, Active Cases of Tuberculosis.	
		Number.	Percentage.	Number.	Per centage.
Metropolitan ..	180,711	6012	3·3	171	0·094
Towns over 5000 population	23,677	335	1·4	21	0·089
Towns under 5000 population and rural areas ..	91,277	2047	2·2	70	0·077
Total	295,665	8394	2·8	262	0·089

The table also suggests that survey work may be a little more profitable in terms of active tuberculosis discovered in the metropolitan area than it is in large or small country towns. But the prevalence of previously unknown cases is really remarkably constant in different areas.

When we examine these survey findings by age and sex we find that previously unrecognized tuberculosis is more than twice as common in males as it is in females. In addition, in the male it becomes strikingly commoner with increasing age (Table II), so that while it was necessary to examine almost 3000 teen-age males to find one new case of tuberculosis, we could achieve the same score by examining two or three hundred old men. Taken over the whole age span, one man in 836 has active tuberculosis, apparently without being aware of it.

TABLE II.

Males Examined and New Cases of Tuberculosis Discovered in Compulsory X-ray Survey, March, 1952, to March, 1957, Classified According to Age Group.

Age (Years).	Number Examined.	Cases Discovered.	Ratio.
0 to 14	4117	3	1 : 1372
15 to 19	14,982	5	1 : 2996
20 to 29	31,521	20	1 : 1576
30 to 39	34,937	34	1 : 1028
40 to 49	29,730	50	1 : 595
50 to 59	20,282	21	1 : 963
60 to 69	13,533	37	1 : 366
70 and over	3075	12	1 : 256
All Ages	152,127	182	1 : 836

In women, the condition is distinctly commoner in the teen-age group, but much rarer in the elderly (Table III); and as I have indicated its prevalence in women is less than half that in men, so that we have had to examine 1794 women for each new case discovered.

It would be of great practical importance to know at what time interval a population should be resurveyed. As yet we have no experience of second compulsory surveys, but after a survey the total new notifications from areas surveyed appear to remain below pre-survey levels for at least two to three years. The results of an initial survey thus form valuable evidence of the prevalence of tuberculosis. The need for further surveys depends on the incidence of tuberculosis in the population concerned, that is, on the number of new cases occurring per year. New notifications from all sources each year give the most valuable over-all indication of incidence.

Figure V shows the numbers of new notifications of pulmonary tuberculosis for each of the past six years, according to age at the time of notification and sex. The first thing that strikes one about these curves is their uniformity in contrast with the curves of death rate. There was a slight decrease in notifications in 1956, and this was most noticeable in teen-age girls and middle-aged men.

The characteristic pattern is that up to puberty the numbers of cases in the two sexes are equal. In the teens notifications of female patients predominate; but after the age of 20 years the excess of male over female cases increases throughout the rest of life.

TABLE III.

Females Examined and New Cases of Tuberculosis Discovered in Compulsory X-ray Surveys, March, 1952, to March, 1957, Classified According to Age Group.

Age (Years).	Number Examined.	Cases Discovered.	Ratio.
0 to 14	4163	1	1 : 4163
15 to 19	13,772	8	1 : 1721
20 to 29	28,021	13	1 : 2155
30 to 39	33,411	22	1 : 1519
40 to 49	27,316	12	1 : 2276
50 to 59	19,707	13	1 : 1516
60 to 69	14,514	8	1 : 1814
70 and over	2634	3	1 : 878
All Ages	143,538	80	1 : 1794

I should emphasize that, unlike the mortality curves, these curves of notifications do not represent rates, but show the actual numbers of notifications. If rate curves were available they would show a much less pronounced hump in early adult life, and the curves would remain distinctly higher in later years.

In endeavouring to relate these curves of notifications to the actual incidence of pulmonary tuberculosis, several factors must be considered. First, there has been a 16% population increase in South Australia in the past six years; and in addition there has been some increase in the intensity of case-finding attempts in that period. Chest clinic attendances have increased by 8%, and the numbers examined in X-ray surveys increased greatly up to 1953 and again in 1956. Tuberculin surveys of school children and National Service trainees began in 1952, and these sources have added a small contribution to the numbers of notified cases. These factors of increased population and increased attempts at case-finding might have been expected to produce increased notifications if the incidence of tuberculosis had remained constant. In fact, the level of notifications has remained almost constant, and it is fair to conclude that the incidence of pulmonary tuberculosis per 100,000 of population is declining. However, from the practical point of view, the constant level of notifications presents us with an undiminished load of new patients each year.

Another important question is whether patients are now being discovered while the disease is at an earlier or less extensive stage. The extent or stage of the disease, as defined by the American National Tuberculosis Association, is stated on the notification form; and the criteria for assigning each patient to the categories of minimal, moderately advanced or far advanced are printed inside the cover of the book of notification forms. There is reason to believe that these criteria are not always closely followed, and there seems to be a tendency to assign patients who are clinically ill or who have a long history

to the far advanced stage, and those with few or brief symptoms to the minimal stage.

However, there has been a slight but progressive decrease each year since 1953 in the percentage of patients notified with far advanced disease—from 13% in 1953 to 9% in 1956. This suggests that the disease is being discovered at an earlier stage now, but the difference over the four-year period is not statistically significant.

Perhaps the proportional decrease in far advanced cases may be due to the discovery of more early cases in X-ray surveys. In fact, in the four years 11·4% of all notifications have been in the far advanced category, while only 8·8% of patients discovered by X-ray survey have been in this group; but again the difference is not large enough in this series to be statistically significant.

Two groups in the population require special mention: recent migrants and aboriginal natives.

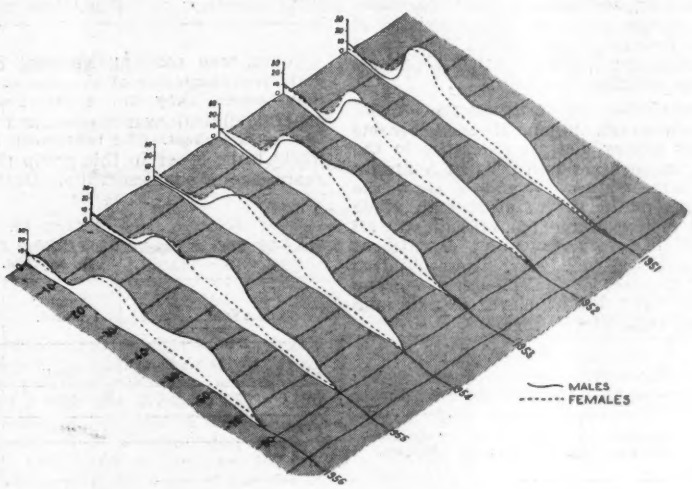

FIGURE V.
Notifications of tuberculosis according to age and sex, 1951 to 1956.

During the past four years we have kept records of new notifications of the disease in migrants who, at the time of notification, have been in this country for less than five years. Of these people 144 have been notified, and they have made up 11·3% of all the notifications in that time. It is difficult to obtain accurate figures for the number of migrants of less than five years' standing who are resident in the State at any time. There is, of course, no check on interstate movements, but the best estimate I can obtain indicates that during the past four years the average proportion of the population who had migrated within the previous five years was 6·5%.

If there were no difference in the incidence of tuberculosis between recent migrants and the remainder of the population, and if the age and sex distribution of the groups was comparable, one would expect that 6·5% of all newly notified patients would be migrants. The difference between the expected rate of 6·5% and the observed rate of 11·3% is statistically significant in groups of this size.

I am sure there is no need to consider the possible role of racial or genetic differences in accounting for this difference in incidence. There are two environmental factors of major importance, which I believe afford the whole explanation. The first is the much higher infection rate, as judged by Mantoux testing, in the migrant population; this will be discussed later. The second is the common practice among migrants of working quite unreasonably hard, for absurdly long hours and with completely inadequate rest, in an effort to become rapidly and firmly established in their new country. I have seen several migrant patients who were working two shifts each week-day in two separate jobs, and who spent Saturday and Sunday building their own homes. In selecting European migrants, it would be unreasonable to exclude all those with evidence of healed tuberculosis. But it is little wonder if, under the circumstances I have described, some of these apparently healed lesions break down.

Notification of new patients from the aboriginal population was rare until the last two years. There were two cases in 1952, three in 1953, two in 1954, 11 in 1955, and 19 in 1956. There is no reason to believe that this increase represents any change in the incidence of tuberculosis in the native population; it follows much more serious attempts at case finding in this group, by both X-ray and tuberculin surveys, and better medical attention for many groups of natives. During the past three years, X-ray surveys have covered the State aboriginal settlements at Point McLeay and Point Pearce, and also a number of aboriginal missions, including Koonibba on Eyre Peninsula and Gerard mission in the Upper Murray River area. Groups of natives in more remote areas, such as Coober Pedy and Talawan (about 100 miles west of Fowler's Bay), were first screened by the Mantoux test, and those with positive reactions were transported to the nearest X-ray unit; while in 1952 almost 400 natives were given Mantoux tests at Ernabella mission station by a team from the Commonwealth Health Department.

There are estimated to be 3000 persons of predominantly or wholly aboriginal blood in South Australia. Approximately 1000 of these have been examined in X-ray surveys, and a further 1261 in tuberculin surveys.

At Point Pearce, Point McLeay and Koonibba missions, the natives were living in wood or stone and iron dwellings, many of which were in poor condition, and in the main the menfolk were engaged in labouring or farm work. At Coober Pedy, Talawan and Ernabella, life was proceeding under semi-tribal conditions. The numbers in these two groups were approximately equal. Six new tuberculous patients were notified as a direct result of these surveys, and only one of these was living under semi-tribal conditions.

These survey findings, together with the numbers of notifications in recent years, indicate that tuberculosis is rather more prevalent in the native population than among white people. However, the disease is very rare among those living under semi-tribal conditions.

Infection Rates.

Let us now turn to the third type of data which the epidemiologist uses in assessing the impact of tuberculosis on the community. It is, of course, the extent to which various groups have been infected with the tubercle bacillus, as revealed by the tuberculin test. We employ the Mantoux test, using 0·1 millilitre of 1 in 1000 old tuberculin (10 tuberculin units).

Certain special groups have been examined, including the contacts of known tuberculous patients, medical students and nurses, and all new patients attending the Adelaide Children's Hospital and the Chest Clinic. There is much valuable information to be obtained from a study of the tuberculin reactor rates in these groups, but they are special groups, and, by reason of their employment or their personal or family history, they are not representative samples of the general population.

During the past five years over 47,000 tuberculin tests have been performed by a trained nurse in the Department of Public Health. The groups examined have consisted of 11,763 National Service trainees, 16,005 school children of all ages in country districts and 19,544 seventh-grade school children in the Adelaide metropolitan area.

In 1952, when examination of metropolitan school children in the seventh grade began, it soon became apparent that there was a very distinct difference in the tuberculin reactor rate between children born in Australia and those born elsewhere. Tables IV and V show the results of a five-year tuberculin survey of seventh grade school children in the metropolitan area. Practically all seventh grade children in every education department primary school were examined each year. These children were almost all 12 or 13 years of age, though the group included the occasional very bright eleven-year-old and the dull or handicapped child of 14 years.

TABLE IV.

Tuberculin Tests on Metropolitan Seventh Grade School Children Born in Australia.

Year.	Number Tested.	Positive Reactions.	Percentage Positive.
1952	3014	256	8·5
1953	3084	195	6·3
1954	3451	154	4·5
1955	3800	196	5·2
1956	4351	183	4·1

In the case of children born in Australia, the decline in the reactor rate from 8·5% to 4·1% has been almost smoothly progressive, and the difference between the first and last figures is highly significant in groups of this size.

The numbers of children born in other countries are naturally much smaller, but they have increased each year. The tuberculin reactor rate is constantly about five times as high in these migrant children, and has shown the same progressive fall of 50% in the five-year period. Again, the difference between the 1952 and 1956 rates is statistically highly significant, as is the difference between the rates in Australian-born and migrant children in each year.

The tables for country school children (Tables VI and VII) are different in two respects. First, they do not represent repeated visits to the same localities, but single visits to a separate group of schools each year. The second difference arises from the fact that, because it was not possible to cover the whole State each year, it was decided to test not only seventh grade children but all the children in each school visited.

Therefore, on account of age alone, one would expect the reactor rates to be lower than in the metropolitan group. In fact they are distinctly lower among both Australian-born and migrant groups, and they show the same 50% reduction in reactor rates over the observation period.

Again, the numbers of migrant children are small, but the differences between 1953 and 1956 rates and between the rates for migrant and Australian-born children each year are statistically significant.

TABLE V.

Tuberculin Tests on Metropolitan Seventh Grade School Children Born Outside Australia.

Year.	Number Tested.	Positive Reactions.	Percentage Positive.
1952	221	87	39·4
1953	270	85	31·5
1954	351	88	25·1
1955	425	79	18·6
1956	577	118	20·4

Young men entering National Service training are not truly representative of the age and sex group from which they come. They are a select group, having passed a general clinical examination and a radiological examination of the chest. The tuberculin reactor rate is therefore likely to be lower in this group than in the male 18 to 19 years age group generally. Until late in 1954 migrant

TABLE VI.

Tuberculin Tests on Country School Children Aged Five to Fourteen Years Born in Australia.

Year.	Number Tested.	Positive Reactions.	Percentage Positive.
1953	2281	119	5·2
1954	4926	223	4·5
1955	1348	55	4·1
1956	6374	158	2·5

youths were not required to enter National Service training. The entry of a substantial number of migrants in 1955 may be the reason for the increased reactor rate in that year (Table VIII).

I must digress for a moment to add that in recent years increasing numbers of National Service entrants had previously been vaccinated with BCG at school. They have

TABLE VII.

Tuberculin Tests on Country School Children Aged Five to Fourteen Years Born Outside Australia.

Year.	Number Tested.	Positive Reactions.	Percentage Positive.
1953	118	22	18·6
1954	321	46	14·3
1955	166	15	9·0
1956	471	38	8·1

been excluded from this table. There has been much discussion of the disadvantages of BCG in robbing the clinician of the tuberculin test as a diagnostic weapon, and in preventing the sort of epidemiological assessment of the situation which I have been describing. Since 1952 we have been offering BCG to non-reactors to 10 units of tuberculin among National Service trainees, seventh grade metropolitan school children and fifth, sixth and seventh grade children in those country schools which we have been able to visit (because we did not expect to return to the same centres within three years).

The Medical Research Council investigation in Britain showed that the incidence of tuberculosis in a BCG-vaccinated group of adolescents was only one-fifth of that in a control group of non-reactors who were not vaccinated. To achieve an 82% reduction in the incidence of tuberculosis, even for a few years, the loss of the diagnostic value of the tuberculin test seems a small price to pay. I am sorry that we have at the same time lost part of the value of the Mantoux test as an epidemiological weapon.

We can still observe the changes in reactor rates at a given age year by year. The use of BCG among the general community is occurring at the age of 12 years each year in the metropolitan area. In the country it is being given to children aged 10, 11 and 12 years not more frequently than every three years. Therefore, we have at all times a fresh group of unvaccinated children whose examination provides the yardstick of tuberculous infection in the community. However, we do not know the rate at which infection occurs after the age of 12 years, and, because we began vaccinating twelve-year-olds in 1952, we cannot hope to find this from examination of National Service trainees in the future.

TABLE VIII.
Tuberculin Tests on National Service Trainees.

Year.	Number Tested.	Positive Reactions.	Percentage Positive.
1952	2212	469	21·2
1953	2270	363	16·0
1954	2349	328	13·9
1955	2461	473	19·2
1956	2471	377	15·3

The final group whose Mantoux reactions have provided interesting information are the aboriginal natives whom I described earlier. The tuberculin reactor rate in native children under 10 years of age was 2·8% in 555 examinations. This is very similar to the rate in white children born in Australia. In the 10 to 19 years age group the rate was 12·9%—again very close to the rate for white Australians; while for those over 20 years of age the rate was 28·4%. It was noteworthy that there were no positive reactors under the age of 15 years in the Koonibba mission natives. At Talawan, among 135 tests, there were only two positive reactions, both in adults. There was no over-all difference in reactor rates between natives living in houses and those living under semi-tribal conditions, but figures given earlier showed that, despite this similar risk of exposure, the environment appears to make a significant difference to the disease attack rate.

There are many points of interest in these tuberculin reactor rates. The outstanding one is the extraordinary reduction in reactor rate in both Australian-born and migrant children in the past five years. Among the migrants this suggests that children reaching seventh grade have now spent a larger proportion of their lives in Australia than had those reaching seventh grade in earlier years.

In 1956, 15% of National Servicemen reacted to tuberculin, while only 4% of seventh grade children did so. We cannot conclude from these figures that the risk of infection rises substantially in the period after leaving primary school. The National Serviceman of 1956 was probably in seventh grade in 1950 or 1951. Our earliest rate for this group is for 1952, when it was 10·6%. When rates are changing as rapidly as these, care is needed in drawing true comparisons.

The strikingly low reactor rate among aborigines on Eyre Peninsula contrasts sharply with those in the far north and in the more civilized areas.

Conclusions.

It is tempting, and I believe necessary, not only to draw conclusions, but also to try to make predictions based on the facts I have presented.

Mortality trends suggest that, while environmental factors and host-parasite relationships have long been reducing the importance of tuberculosis as a cause of death, treatment has made its first significant contribution in the past 10 years. The lower mortality is accompanied by some increase in respiratory invalidism in elderly males.

One consequence of these changes is a change in the type of hospital facilities needed for the care of the tuberculous. The old-style sanatorium, where those fortunate enough to survive the waiting list spent years taking the cure and perhaps learning to live with their disease, is not the appropriate institution today. We need, on the one hand, active chest units or chest hospitals where relatively brief and intensive in-patient medical and surgical investigation and treatment are given by high quality staff, who subsequently continue the care of these patients as out-patients. On the other hand, there is need for in-patient and out-patient care of partial and total respiratory cripples—almost all males—from pulmonary fibrosis, bronchitis and emphysema.

The information on the prevalence of tuberculosis in South Australia suggests that the sooner we complete our X-ray survey of the whole adolescent and adult population the better, because for every 1100 people not yet examined there is one unknown sufferer from active tuberculosis moving in the community who is not under treatment.

It is also apparent that total population surveys should be repeated at intervals, and the appropriate interval appears to be three years.

Incidence figures suggest that we will need to maintain something like our present numbers of beds for tuberculous patients for some time, but that we are not likely to need more beds, despite the rising population.

One special feature in recent years has been the more active treatment of children with clinically active primary disease, and the treatment of newly infected children, even in the absence of clinical disease. There is no doubt at all that these activities have contributed to the decreased incidence of tuberculous meningitis; but, at the same time, they have entailed the admission of many more children with primary disease and fresh infection to hospital, so that special provision is being made for their care.

The spectacular changes in tuberculin reactor rates in recent years are the best index of the decreasing impact of tuberculosis on the community. Those engaged in tuberculosis work can take more encouragement from these changes than from any others I have mentioned. One consequence of this has been that although primary disease in children still occurs too frequently it is much rarer than it was; but at the same time primary disease in adolescents and young adults has become relatively more common. We have long been aware of the hazards of primary disease in early childhood, and of the relatively benign nature of this condition during the primary school years.

Proper treatment of infectious patients and the examination of most pregnant women for evidence of active tuberculosis have done much to reduce primary disease in infancy. It is in the adolescent group who are moving further from the family circle that we are faced with a comparatively new problem in primary tuberculosis. Primary disease occurring in an adolescent appears much more likely to progress to destructive phthisis or to cause widespread dissemination than does the same condition in a primary school child. It is therefore in the teenage group that we may expect the greatest benefit from the widespread use of BCG vaccine, and it is here that we are using it.

I have described a number of observations and suggested explanations for some of them, and I have touched on lines of action which have been planned with these observations in mind.

The following are certain obvious unanswered questions of a practical nature. How can spreaders of tuberculous infection be detected sooner? How can we deal more effectively with the organisms themselves? How can we

obtain more complete epidemiological information? There are more fundamental questions, too, in the realm of pathology. To me, the two important ones are: What are the factors which determine the change from tuberculous infection to tuberculous disease? Why do men get emphysema?

The answers to these questions would help towards the completion of one of the tasks which Laennec began when he put chest medicine on a rational basis.

THE MALABSORPTION SYNDROME.[1]

By D. W. Piper,
Sydney.

A defect in absorption by the gastro-intestinal tract may be in respect of a single substance or may be in respect of many dietary constituents. Typical of the single absorptive defect is that present in pernicious anæmia, in which lack of secretion of the intrinsic factor by the stomach results in defective absorption of vitamin B_{12}. The multiple absorptive defects are characterized by steatorrhœa, and it is preferable to use the term steatorrhœa when the broad malabsorptive syndromes are meant. The term steatorrhœa was applied by Kuntzmann in 1824 to the presence of visible fat in the fæces, but is now used to signify excess fat in the fæces as determined by biochemical analysis.

The Physiology of Fat Absorption.

Before fat can be absorbed it must be partially digested in the gastro-intestinal tract. The details of fat digestion and absorption have been the subject of much debate and research. The fat is emulsified by the bile salts aided by the presence of fatty acids and monoglycerides formed from triglycerides previously hydrolysed; the emulsion formed consists of particles sufficiently small to allow a large surface for the action of lipase and to permit particulate absorption. The controversy regarding fat absorption centres around how far lipolysis progresses before absorption takes place and what proportion is absorbed via the lacteals and what proportion by the portal blood. Frazer (1952) incorporated both of these pathways in his partition hypothesis. According to him lipolysis is incomplete, 70% of the absorbed fat being unhydrolysed; the emulsified particles of less than 0·5 micron are absorbed by the lacteals and pass via the thoracic duct into the venous blood; under normal circumstances most of the saturated long chain fats and probably most of the unsaturated long chain fatty acids are absorbed by this route. Other fatty acids, especially short chain fatty acids, form a water-soluble fatty acid-bile salt complex with the bile salts which are absorbed by the portal blood and carried to the liver. However, other investigators have demonstrated that lipolysis is more complete, one-third of the absorbed fat being completely hydrolysed and most of the remainder existing at the monoglyceride level (Reiser, 1955). Experiments with isotope techniques suggest that over 90% of the triglyceride and fatty acid is absorbed by the lymph (Borgstrom, 1951; Bloom *et alii*, 1950). Kiyasu and his colleagues (1952) have demonstrated that the majority of long chain fatty acids are transported via intestinal lymph as triglycerides, and fatty acids of shorter length are carried in the portal blood as free fatty acids.

Origin of Fæcal Fat.

The fat in normal fæces is partially of non-dietary and partially of dietary origin (Frazer, 1955). Most of the endogenous fat appears to originate from desquamated intestinal epithelium, and a small amount arises from bacteria and sterol excretion in the bile (Pessoa, Kim and Ivy, 1953). When the fæcal fat content is increased above the normal range, the excess may be of dietary or non-dietary origin; for this reason many workers believe it is incorrect to convert fæcal fat excretion into any coefficient of fat absorption, but to express the fæcal fat analysis on the factual basis of grammes of fæcal fat per day (Frazer, 1955). Wollaeger, Comfort and Osterberg (1947) have collected together many of the recorded observations from the literature and have shown that as the fat intake increases in normal subjects there is a significant rise in total fat output, the mean excretion rising from four grammes with an intake of 50 grammes to six grammes with an intake of 150 grammes. Using five-day fat balances and three different intakes between 60 and 150 grammes, it has been demonstrated that 95% of normal people will excrete less than seven grammes of fat per day (Annegers, Boutwell and Ivy, 1948). In clinical work a three-day fat balance is adequate, the patient being on a known fat intake of 75 to 100 grammes for three days before and during the period of collection. The quantitative method of van de Kamer *et alii* (1949) has greatly simplified the analysis, and this method requires about one hour to perform an estimation. On a diet of 75 to 100 grammes, seven grammes per day is probably the upper limit of normal; over 10 grammes per day is certainly abnormal. Cases with borderline results (excretion of seven to 10 grammes) or in which the bowels are constipated require further study by more prolonged fat balances. The estimation of the ratio of split to unsplit fat in the fæces is of little value, because hydrolysis may be produced by intestinal bacteria in the absence of pancreatic secretion and lipolysis continues during and after collection, producing a falsely high degree of splitting (Cooke *et alii*, 1946).

Microscopic examination of the fæces for fat globules, analysis of single fæcal specimens for its percentage fat content, vitamin A absorption tests and radiological examination of the small intestine for the so-called flocculation pattern, are all liable to so many inaccuracies as to have no place in the diagnosis of steatorrhœa.

Classification of Steatorrhœas.

Ætiologically and clinically there are three groups:

1. Enterogenous, in which abnormalities of the small intestine result in a generalized absorptive defect.

2. Pancreatic, in which deficient secretion of lipase and trypsin results in defective absorption of fat and to a lesser extent of protein.

3. Hepatic, in which lack of bile salts causes defective absorption of fat.

Enterogenous Steatorrhœa.

In this group there is defective absorption of almost all dietary constituents, though not all are involved to an equal extent. This is well exemplified after extensive surgical removal of the small intestine, where fat absorption is the most impaired, protein absorption less impaired and carbohydrate little impaired, if at all (Althausen, Uyeyama and Simpson, 1949).

Small bowel steatorrhœas may be classified as follows.

1. Primary, which are cœliac disease, idiopathic steatorrhœa and tropical sprue.

2. Secondary to organic changes in the small bowel, such as regional ileitis, tuberculous enteritis, Whipple's disease, scleroderma, *Giardia lamblia* infestation, agammaglobulinæmia, reticulosis, amyloid disease, ileitis secondary to ulcerative colitis, gastro-colic or ileo-colic fistulæ following gastrectomy, intestinal strictures, blind loops and jejunal diverticulosis, following extensive surgical removal of the small intestine, and congenital malformations of the intestinal lymphatics.

The symptomatology is best thought of relevant to the multiple absorptive defects involved.

1. Fat, protein and carbohydrate: Defective absorption of fat gives rise to diarrhœa, with the typical pale, frothy, offensive stools. The diarrhœa typically occurs in attacks several times each year. Amino acid loss, though increased, is not great, the three to four gramme nitrogen excretion being equivalent to 18 to 24 grammes of protein lost each day. Impaired carbohydrate absorption gives rise to flatulence and the typical flat glucose tolerance curve.

[1] Read at a meeting of the Section of Medicine, British Medical Association, on October 10, 1957.

ILLUSTRATIONS TO THE ARTICLE BY K. VINER SMITH.

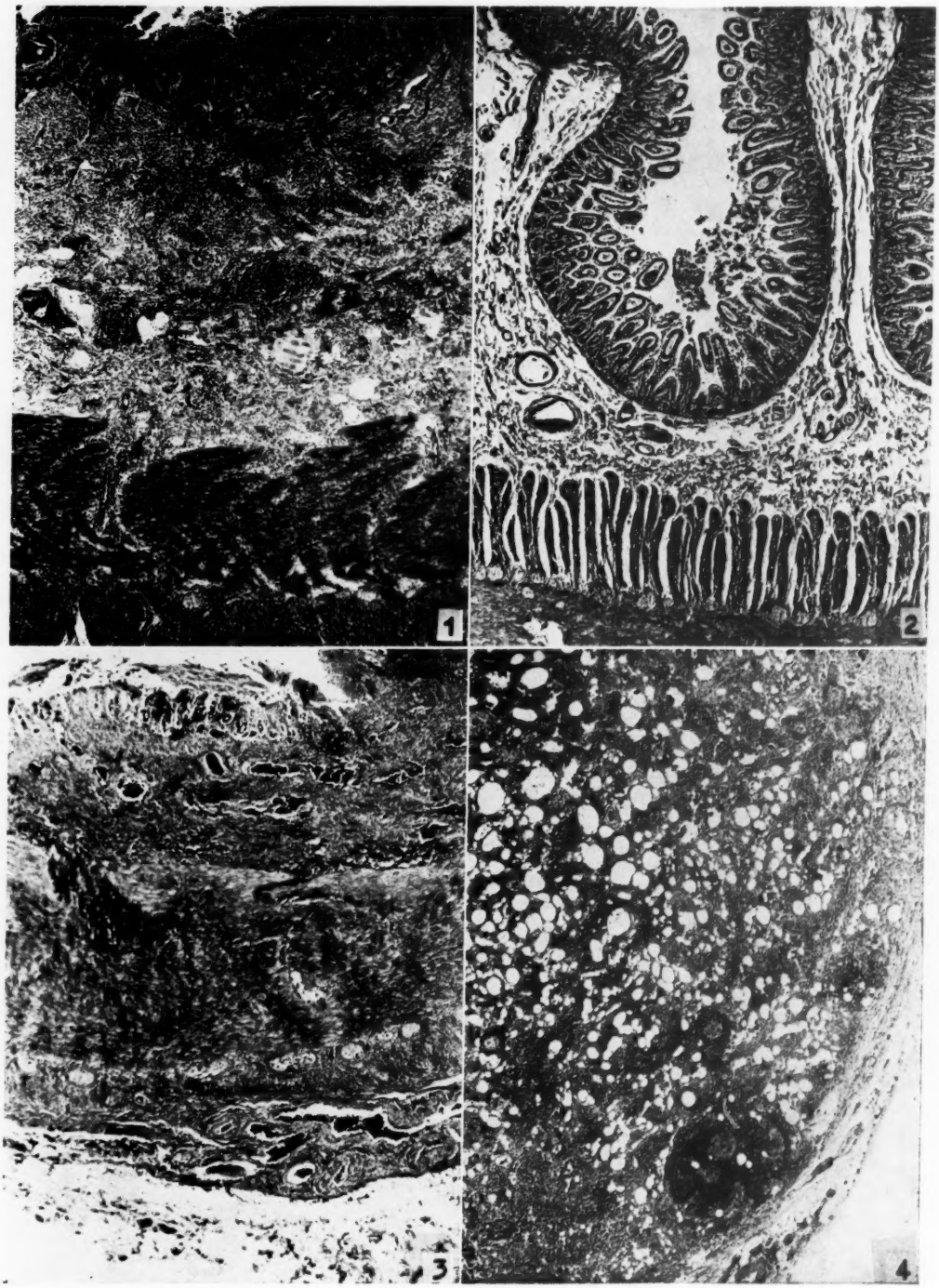

FIGURE I: Small intestine in regional ileitis. The mucosa and submucosa are thickened and there has been much fibrosis. (× 25.) FIGURE II: Normal small intestine for comparison. (× 25.) FIGURE III: Small intestine which has been damaged by irradiation. The mucosa and submucosa, which occupy the upper quarter of the photograph, are densely fibrosed. The blood vessels in these coats and in the subserous layer are unusually large. (× 25.) FIGURE IV: Mesenteric lymph node in Whipple's disease. The spaces indicate the sites from which fat has been dissolved. (× 25.)

ILLUSTRATION TO THE ARTICLE BY GEORG BERCI.

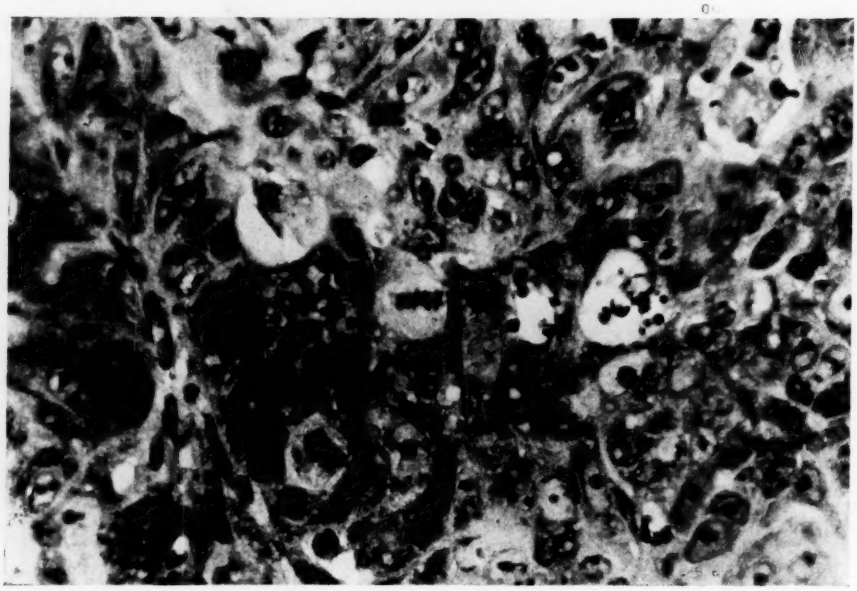

FIGURE III.

Metastasis in the liver in a case of carcinoma of the caecum, six months after hemicolectomy. (Hæmatoxylin and eosin stain, × 270.)

ILLUSTRATIONS TO THE ARTICLE BY GEORGE READ.

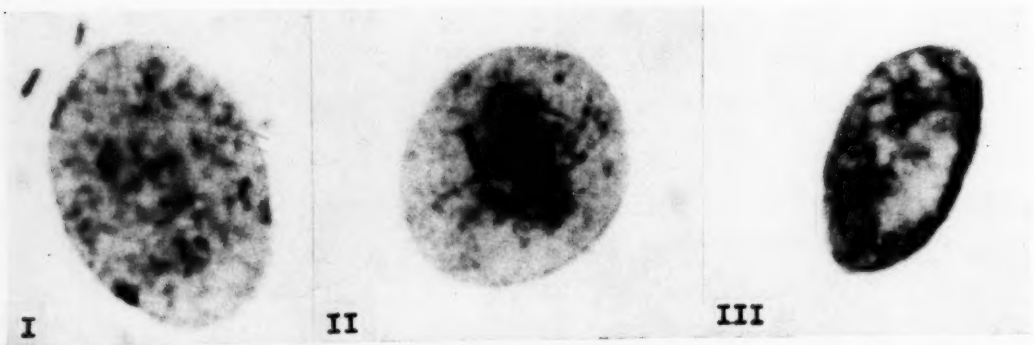

FIGURE I: Female buccal mucosal smear cell showing the stained chromatin spot at 7 o'clock. FIGURE II: Male buccal epithelial cell with no such characteristic spot. FIGURE III: An indeterminate buccal cell with gross bacterial contamination. (× 3600.)

Most of the protein depletion is related to defective carbohydrate absorption; there is not an adequate amount of carbohydrate absorbed immediately post-prandially to provide the high energy phosphate bonds necessary to convert the amino acids into liver protein. This defect, combined with the protein loss in the fæces, produces the weight loss, lassitude, hypoalbuminæmia, osteoporosis and œdema that dominate the symptomatology of steatorrhœa.

2. Minerals: Balance studies show defective absorption of all electrolytes, especially during the exacerbations of the disease (Comfort *et alii*, 1953). The defective absorption of iron results in an iron deficiency anæmia (Badenoch and Callender, 1954), and loss of potassium to weakness, apathy and, when extreme, death. Loss of calcium partially causes cramps, tetany and osteomalacia, and sodium loss may be responsible for lassitude and hypotension. Defective water absorption results in nocturnal polyuria and impaired water diuresis and, in crises, dehydration.

3. Vitamins: In the enterogenous group both water and fat soluble vitamins are involved. Defective absorption of vitamin B_{12} gives rise to megaloblastic anæmia, glossitis, subacute combined degeneration and peripheral neuritis, and defective absorption of folic acid gives rise to glossitis and megaloblastic anæmia. Loss of vitamin D causes osteomalacia, tetany and cramps, and loss of vitamin K gives rise to hæmorrhagic tendency. Vitamins involved to a lesser extent include nicotinic acid, ascorbic acid and riboflavine.

Other symptoms of which the cause is not obvious include pigmentation, clubbing of the fingers and mild fever.

Enterogenous steatorrhœa is most commonly of idiopathic origin. In such cases the patients usually have a long history of diarrhœa and 43% have a history of cœliac disease in childhood (Cooke, Peeney and Hawkins, 1953). In an analysis of 100 cases, Cooke *et alii* found that lassitude, loss of weight, diarrhœa, glossitis and flatulence were the most common symptoms, and anæmia was the most common sign. Its diagnosis is essentially one of exclusion, and many cases that are thought to be idiopathic after detailed investigation are found by autopsy or by follow-up studies to be due to one of the secondary causes listed; it must be excluded in all cases of obscure anæmia, protein depletion and unexplained diarrhœa and weight loss. Finally, in 20% of all cases of idiopathic steatorrhœa the patients have no bowel symptoms.

It is not possible to describe all the enterogenous steatorrhœas, but brief mention will be made of a few.

Tropical Sprue.

Clinically, tropical sprue differs from idiopathic steatorrhœa in its geographical distribution, the high incidence of megaloblastic anæmia, the rarity of hypocalcæmic and hypoprothrombinæmic symptoms and the dramatic response to folic acid and the broad spectrum antibiotics. Though these distinguishing features are frequently quoted, it is probable, as Professor Woodruff (1952) has pointed out, that the only difference between idiopathic and tropical sprue lies in the duration of the disease, for idiopathic steatorrhœa is a disease that persists with remissions and exacerbations for the remainder of the patient's life, and tropical sprue is a disease which with proper treatment can be cured. There is no evidence that folic acid will restore to normal defective fat absorption (Woodruff, 1949, 1952), and it is likely that the dramatic responses obtained with folic acid by the Caribbean investigators were due to the fact that they were confusing cases of nutritional megaloblastic anæmia with tropical sprue; in their diagnosis they laid emphasis on the presence of megaloblastic anæmia and not on biochemical evidence of steatorrhœa, as do English and Indian workers.

Steatorrhœa Associated with Intestinal Strictures, Fistulæ, Loops and Diverticulæ.

The association of steatorrhœa and megaloblastic anæmia with intestinal strictures, loops and fistulæ has been demonstrated experimentally and clinically. The observation of Renshaw *et alii* (1946) that in dogs with gastro-colic fistulæ the stomach contents did not pass into the colon, but that the colon contents readily entered the stomach, led to the hypothesis that the maifestations of the syndrome were due to contamination of the stomach and small intestine with colonic bacteria rather than to the direct passage of food from the stomach to the colon. In this group of conditions as well as in the more recently documented syndrome of jejunal diverticulosis (Dick, 1955), the steatorrhœa is often mild, acid is present in the stomach and the anæmia is megaloblastic; Halstead (1956) has demonstrated defective absorption of vitamin B_{12} which can be corrected by the administration of aureomycin but not by intrinsic factor. Table I compares pernicious anæmia, idiopathic steatorrhœa, intestinal strictures, loops and diverticulæ, nutritional megaloblastic anæmia and tropical sprue, as regards vitamin B_{12} absorption, the effect of intrinsic factor and aureomycin, and their response to vitamin B_{12} and folic acid.

Pancreatic Steatorrhœa.

Most patients with pancreatic steatorrhœa have evidence of gross pancreatic disease, such as diabetes, calcification of the pancreas or fatty stools, one of these advanced signs being usually present when fat balance reveals steatorrhœa. Dornberger *et alii* (1948) found that fat and nitrogen loss were normal in ten patients with relapsing pancreatitis without the above-mentioned sequelæ, and that those with more than one of these sequelæ had steatorrhœa roughly proportional to the pancreatic damage as judged clinically. The mean fat loss was found to be 21·6 grammes in the advanced cases, and the nitrogen loss was 3·2 grammes or twice the normal value. As a group, fat loss is greater in pancreatic steatorrhœa than in idiopathic steatorrhœa, but it is not possible to distinguish between these two groups by the extent of the fat and nitrogen loss (Card, 1957). This contrasts strikingly with the statement made by Thaysen in 1926 and still widely quoted that the distinguishing feature of pancreatic steatorrhœa was a high nitrogen excretion, which he claimed was 13 to 18

TABLE I.

Some Characteristics of Certain Types of Enterogenous Steatorrhœa.

Condition.	Vitamin B_{12} Absorption.	Effect of Intrinsic Factor.	Effect of Aureomycin.	Response to Vitamin B_{12}.[1]	Response to Folic Acid.
Pernicious anæmia	Impaired	++++	–	++++	++
Idiopathic steatorrhœa	Impaired	–	–	+++	+++
Intestinal strictures, loops and diverticulæ	Impaired	–	++++	+++	++++
Nutritional megaloblastic anæmia	Normal	–	–	+	++++
Tropical sprue	Impaired	–	++++	++	++++

[1] It is probable that the response to vitamin B_{12} and folic acid applies only to relief of symptoms known to be due to either of these vitamins, i.e., megaloblastic anæmia, glossitis, subacute combined degeneration and peripheral neuritis.

grammes per day. Even in the advanced cases of pancreatitis seen clinically with diabetes, calcification and typical fatty stools, there must still be a considerable amount of pancreatic enzyme secreted, for after total pancreatectomy the fat loss is in the vicinity of 50 grammes, and the nitrogen loss varies from four to eight grammes per day (Waugh *et alii*, 1946).

Before steatorrhœa becomes clinically and biochemically manifest, pancreatic disease may reveal itself by episodes of abdominal pain typical of chronic relapsing pancreatitis, and at this stage special investigations may show abnormalities. The combined secretin-pancreozymin test is the most informative, and it has been shown that amylase secretion may be defective when the volume and bicarbonate response are within the normal range (Marks and Thompsett, 1957). Table II contrasts the features of pancreatic and idiopathic steatorrhœa.

Table II.

The Features of Idiopathic and Pancreatic Steatorrhœa.

—	Idiopathic Steatorrhœa.	Pancreatic Steatorrhœa.
History	Cœliac disease in 40% to 50%.	Pain, diabetes, calcification.
Anæmia	Very common, often megaloblastic.	Rare; if present, usually iron deficiency.
Glossitis	Common.	Rare.
Glucose tolerance curve	Flat.	Normal or diabetic.
Pancreatic secretion (secretin-pancreozymin test)	Normal.	Deficient response as regards amylase, bicarbonate and volume.
Fat loss	Increased.	Increased.
Nitrogen loss	Increased.	Increased.
Vitamin deficiencies	Common.	Rare.
Electrolyte depletion	Common.	Rare.

Hepatic Steatorrhœa.

Bile salts are necessary for the emulsification of triglycerides and the absorption of fatty acids; it is probable that the steatorrhœa in hepatocellular and obstructive liver disease is due to defective fatty acid absorption rather than to impaired triglyceride emulsification, because it is known that triglyceride digestion can be complete in the absence of bile (Annegers, 1954). Gross *et alii* (1950) studied the fat and nitrogen loss in parenchymatous liver disease and found that the fæcal fat content averaged 13 grammes per day on an intake of 101·6 grammes, but the nitrogen loss was always normal. The fat excretion was more abnormal when studied during the periods of active disease, tending to diminish as recovery progressed. The absence of azotorrhœa in steatorrhœa due to obstructive jaundice and hepatocellular disease is in contrast to the findings in the pancreatic and enterogenous steatorrhœas, in which excess fat loss is always accompanied by excess nitrogen excretion.

In those cases of steatorrhœa due to obstructive jaundice, the incidence of bone disease, either osteoporosis or osteomalacia, is common if the condition has persisted for more than two years (Atkinson *et alii*, 1956). Evidence of malabsorption of water, glucose, minerals and water soluble vitamins is usually absent.

Summary.

The physiology of fat absorption and methods of estimation of fat excretion are discussed.

A classification of the malabsorption syndrome is presented, and an attempt is made to define the varying syndromes which have the common factor of steatorrhœa.

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THE MALABSORPTION SYNDROME.[1]

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IN this brief address I intend to describe and demonstrate the anatomical changes in some of the diseases which give rise to the malabsorption syndrome; and to consider what help can be gained in understanding the condition by a study of these changes. I am taking the term "malabsorption syndrome" to mean a condition in which fats are incompletely absorbed, leading to steatorrhœa. This is always accompanied by failure to absorb some vitamins, minerals, carbohydrate and possibly some protein.

Normal Digestion and Absorption of Fats.

The normal digestion and absorption of fats occur in the small bowel. When the food, having been churned up in the stomach, passes into the duodenum, bile and pancreatic juices are added to it. The biochemists are divided on the part these two substances play. One school considers that only a small proportion of fat is split by pancreatic lipase into fatty acids and glycerides and that the remainder of the neutral fat is finely emulsified with the aid of these breakdown products and of bile. It can then be absorbed by the intestine. The other school holds that much more fat is split by the lipase, and that the main functions of the bile are to emulsify the fat and, later, to form soluble compounds with the fatty acids, which are thus easily absorbed. Whichever school of thought is correct, it is agreed that pancreatic lipase and bile are both necessary for normal fat absorption (Cantarow and Trumper, 1955).

When the fat has passed through the intestinal epithelium it reaches the lymphatics in the mucosa. In these vessels it passes to the mesenteric lymph nodes and filters through them. Eventually it flows into the thoracic duct and from there into the blood stream.

Failure of Fat Absorption.

In a large number of diseases there is a failure in fat absorption because of a breakdown in the normal process at one or more points. Bile or pancreatic lipase may fail to reach the duodenum, either because the liver and pancreas do not form the substances or because the ducts are blocked. The wall of the intestine may fail to take up the fat and to pass it on to the lymphatics. And finally the lymphatics may become blocked. The diseases are best classified under the following four headings: (i) absence of bile in the duodenum; (ii) absence of pancreatic lipase in the duodenum; (iii) failure of absorption by the small intestine; (iv) obstruction of lymphatics.

In considering the malabsorption syndrome we can disregard diseases of short duration as in these the steatorrhœa does no significant harm to the patient.

Absence of Bile.

In the first group of diseases the bile may be prevented from reaching the duodenum because of stone, tumour, or more rarely, enlarged lymph nodes obstructing the duct. There is some loss of fat in the stools, but it is never great and is usually negligible. Only when the obstruction persists for a long time does the steatorrhœa assume importance.

If the secretion of bile ceases because of disease of the liver cells, the resulting steatorrhœa is unimportant compared with the other effects.

Absence of Pancreatic Lipase.

Obstruction of the pancreatic duct by calculus or tumour leads to the loss of a great deal of ingested fat, as the amount of lipase secreted by the wall of the intestine is insignificant. The pancreas shrinks because the acinar cells atrophy and disappear. The organ is finally reduced to a collection of islets of Langerhans and some dilated ducts in fibrous tissue. This was the phenomenon used by Banting and Best to isolate insulin. Fibrocystic disease of the pancreas is now generally considered to result from an increased viscosity of its secretions, which obstruct the ducts (Bodian, 1952). The ducts become dilated, but not cystic, and contain inspissated secretion. Most of the acini are lost, but the islets remain. There is a great increase in fibrous tissue. As a result of the loss of pancreatic secretions, about half the ingested fat of an ordinary diet is not absorbed (Anderson, 1953).

Failure of the pancreas to secrete may occur from acute or chronic pancreatitis. The acute form is important only when it so damages the organ that its function is permanently impaired. Chronic pancreatitis is a term which includes both relapsing inflammation and fibrosis of the pancreas of unknown cause. In either disease both the exocrine and the endocrine glandular tissue may be damaged to such an extent that steatorrhœa and diabetes develop. The pancreas is small, hard and nodular.

Failure of the Small Intestine to Absorb.

The small bowel may be unable to perform its part in the absorption of fats under a number of conditions. Extensive resection is such a one, although it is remarkable how efficient a few remaining feet of bowel can be. Samson Wright (1952) describes a patient who had had all but the upper three feet of the small intestine removed, yet he absorbed 80 grammes of fat out of 200 grammes ingested.

Gastro-colic fistula and partial small bowel obstruction may lead to steatorrhœa. The abnormal bacterial flora which develop are thought to be the main cause (Renshaw, Templeton and Kiskaddon, 1946).

Regional ileitis, if extensive, may cause deficient absorption. The widespread inflammation of the submucous and mucous layers with subsequent fibrosis will hinder the passage of fats from the bowel lumen to the lymphatics and will obstruct some lymphatics. The disease is more extensive than naked eye inspection suggests, and the so-called "skip areas" are often seen on microscopic examination to be affected.

Steatorrhœa was recently observed in a woman aged 45 years. She had had abdominal pains for three years. After the steatorrhœa had persisted for 10 months, laparotomy was performed. The surgeon reported that her ileum resembled a red hose pipe. A section from this ileum (Figure I) shows ulceration with fibrosis and non-specific chronic inflammation. Some lymphatics are dilated, indicating obstruction. This is, in my experience, the common histological appearance in regional ileitis. Figure II shows a section of normal ileum for comparison.

Irradiation enteritis is due to deep X-ray treatment of the abdomen and pelvis, which may cause such permanent injury to the small intestine that steatorrhœa results. An early stage of multiple ulceration is followed by fibrosis of the mucosa and submucosa. As an example of this, Figure III shows a section of small intestine. It is from a woman who at the age of 26 years had a carcinoma of the ovary removed and was given a course of deep X-ray treatment. For 18 years after this she had diarrhœa, which was not very severe but was not helped by treatment. She was then admitted to hospital with a carcinoma of the ileo-caecal region. Her fæces contained much fat. At autopsy the whole of the small intestine was found to be congested. The mucosa appeared thickened and red. The photograph demonstrates the extensive fibrosis in the mucous and submucous layers.

Whipple's disease, or intestinal lipodystrophy, is rare. Excessive amounts of a glycoprotein are found in the mucosa, and in the mesenteric nodes there is enlargement and cyst formation due to the presence of fat and glycoprotein (Black-Schaffer, 1947). The lymphatics may be dilated, indicating that the resulting steatorrhœa is partly due to the condition of the bowel wall and partly to lymphatic obstruction. Figure IV shows a mesenteric lymph node from a patient who was suffering from this disease. The spaces represent areas from which the fat has been dissolved in processing.

Cœliac disease, idiopathic steatorrhœa and sprue are accompanied by no characteristic changes in the tissue and there is therefore nothing I can show you. They are most important; but the morbid anatomist can make no real contribution to their understanding.

[1] Read at a meeting of the Section of Medicine of the British Medical Association (New South Wales Branch) on October 10, 1957.

Obstruction of the Lymphatic Flow.

Tuberculosis may cause caseation and fibrosis of mesenteric nodes with obstruction to the lymph flow. If there is tuberculous enteritis as well, this may contribute to the steatorrhœa by hindering absorption.

Tumours of lymphoid tissue, such as Hodgkin's disease, lymphosarcoma and giant follicular lymphoma, in which the architecture is destroyed and the sinuses are blocked, may cause appearance of the malabsorption syndrome.

Congenital maldevelopment of lymphatics may lead to obstruction even in adult life. Such an event was seen in a man aged 48 years who was admitted to hospital with symptoms and signs of sprue, including steatorrhœa. He died of broncho-pneumonia. At autopsy there was an extensive lymphangioma of the mesentery as well as a cartilaginous hamartoma in the lung. It must be assumed that obstruction to the lymphatics had occurred by clotting in the stagnant lymph or possibly by fibrosis.

Kwashiorkor.

Along with many other diseases I have omitted kwashiorkor from the discussion as the sequence of events and the cause of the steatorrhœa are obscure. Recent reports (Davies, 1948; Waterlow and Bras, 1957) suggest that atrophy of acinar cells of the pancreas may be the primary lesion in this disease.

Summary.

In conclusion, it can be repeated that the diseases which give rise to the malabsorption syndrome are those which hinder the digestion and absorption of fat in one of four ways. Bile may fail to appear in the duodenum because of disease in the liver cells or in the bile ducts; lipase from the pancreas may be absent from the duodenum because of failure of the acinar cells or obstruction of the duct; disease of the bowel wall can lead to incomplete absorption of the fats; and a number of pathological conditions may obstruct the lymph flow from the intestinal mucosa to the blood stream.

Acknowledgements.

I wish to thank the Department of Illustration, University of Sydney, for the photographs, and Professor F. R. Magarey for his advice in preparing this paper.

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AIR-DRIVEN DRILL-BIOPSY GUN.

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THE value of biopsy in the diagnosis of many diseases is beyond dispute. Very often biopsy alone will yield a positive diagnosis, when a wide variety of biochemical tests, X-ray investigations and other diagnostic aids have left the diagnosis still unproven. This is especially the

[1] Presently in receipt of a grant from the Rockefeller Foundation.

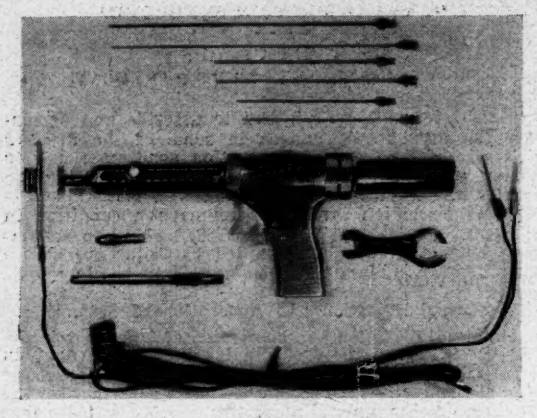

FIGURE I.

The biopsy gun with complete set of attachments. The diameters of the needles available are from 1·4 millimetres to 2·5 millimetres (external diameter). The lengths of the needles vary from 80 to 200 millimetres. The cutting edge of the needle is circular and sharp.

FIGURE II.

Gun during operation. When the trigger is pressed lightly the needle rotates at high speed. If it is then pressed to the limit the needle advances to the predetermined depth, and when the trigger is released the needle immediately returns to its sheath.

case in cancer, where a certain diagnosis is the essential preliminary to rational treatment and a prerequisite in the evaluation of alternative types of therapy.

Open biopsy has much to commend it for it is then possible under direct vision to take an adequate and representative tissue sample, on which the pathologist can reasonably be expected to express a firm opinion. At the same time, however, this may involve an operation of some consequence, which is often undesirable. In an attempt to save the patient the discomfort of a formal operation, a variety of aspiration biopsy techniques have been introduced and are widely practised. Often, however, these methods yield very unsatisfactory tissue fragments and, even when a pathologist is accustomed to handling them, it may be quite impossible for him to make a worthwhile report on such a small sample. There is further risk that the sample may not be representative. In addition, it is often difficult to obtain a satisfactory yield of tissue from a very fibrous tumour, of which a breast scirrhus is one of the commonest examples.

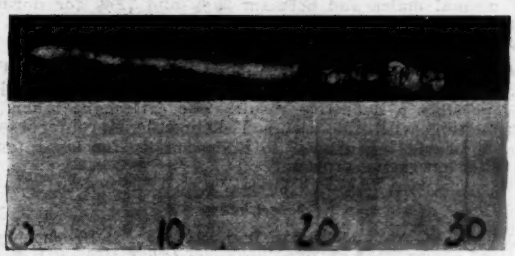

FIGURE IV.

Tissue core from liver. Calibration is in millimetres.

In an attempt to improve the bulk and quality of the yield, Kirschner (1935) introduced an instrument based on the principle of the rapid revolutions of a hollow needle held in a dental handpiece, powered by an electric motor. Ellis, an English radiotherapist, reported in 1947 a wide experience in the use of a similar technique, and in his hands drill biopsy technique seemed to have a wide range of usefulness.

Morrison, in 1951, introduced the gas turbine principle to the drill-biopsy technique. A hollow needle, rotated at almost 20,000 revolutions per minute (driven by compressed air), was advanced by hand, and when the structure for biopsy had been sampled it was withdrawn along the same line.

The drill which we are about to describe has the same basic design but incorporates several novel features which we believe make it a much more valuable instrument, free from some of the disadvantages of earlier methods and involving less risk to the patient. (i) The hollow needle rotates at 10,000 revolutions per minute (driven by compressed air at a pressure of 44 pounds to the square inch). Its advance and return are controlled by the trigger and are entirely automatic. (ii) On the muzzle of the gun is a flat disk, which is pressed against the skin at the biopsy site (the needle is introduced through a small incision made under local anæsthesia with a tenotomy knife). Since the movement of the hand is not transferred to the needle, the operator need concentrate only on maintaining the alignment of the instrument. (iii) The needle advances to a chosen depth, predetermined by a screw adjustment on the barrel of the gun in increments of 6·35 millimetres (one quarter of an inch). (iv) The time taken for the needle to complete its journey can be modified to suit the consistency of the tissue being sampled. (v) All parts of the gun which come into contact with the body can be sterilized. (vi) Attachments make the gun suitable for taking biopsies of the cervix uteri and of bone. The core of tissue is found in the hollow needle after its removal. A good core will measure 20 to 30 millimetres and when mounted will occupy an average low power field.

The tissue shows no sign of burning or of other injury. Our clinical experience with the instrument is not yet considerable, but we are so far encouraged by our success. The track of a biopsy taken percutaneously from the liver just prior to laparotomy has been observed closely after opening the abdomen, but no significant bleeding has so far been encountered.

Acknowledgement.

The gun was entirely constructed in the workshops of the Alfred Hospital, Melbourne, by Mr. Frank Merei.

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SOME CONSIDERATIONS OF GENETIC SEX DETERMINATION.

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IT has not been an easy problem to decide which method of genetic sex determination would prove to be the most practicable. From the point of view of the least inconvenience to the individuals concerned, it was felt that the method using buccal mucosal smears might provide the best answer. Our early interest was an investigation of possible hermaphroditism, pseudohermaphroditism and several cases of transvestism, together with some extensions of the technique to other subjects for genetic sex determination referred to the Department of Obstetrics.

The buccal mucosal smear method of Moore and Barr (1955) was finally chosen, as it was thought that some mothers and babies would be resentful of finger pricking for the leucocyte determination of sex (Davidson and Smith, 1954) or of skin biopsy (Emery and McMillan, 1954). In a critical analysis there did not seem to be much advantage in these alternative methods. The initial approach and some results are presented in this article. The method depends, essentially, on the detection in females of a chromatin mass in the stained nucleus of epithelial cells.

Methods.

A group of 25 physiotherapists in their second year of training, who had declared that they were physically and psychologically normal, was the female volunteer group. These declarations were not questioned. Another group

[1] Research Fellow, King George V and Queen Mary Maternal and Infant Welfare Foundation, aided by grants from the Medical Research Committee of the University of Sydney.

of 25 males, most of whom were confirmed parents of children, formed a comparable unit. After these two groups, one child, aged eight years, and three babies, all of indeterminate sex, were submitted to the test.

The fixing and staining methods of Moore and Barr were used consistently, although a period of 18 hours for fixing was decided upon after various times had been tried. The staining technique was not basically altered. When a magnification of ×1350 was used, it was found at an early stage that the preservation of the slides by mounting them in Canada balsam and by using a coverslip introduced too many refractive errors, so recourse was had to photography in selected cases immediately the slides had been stained. The final standard was the confirmation by individual observations of the presence of the chromatin mass in the stained epithelial cells. Observations were made by two trained microscopists and by the writer. The only instructions were to avoid counting cells with obvious bacterial contamination and to count "reliable" cells. Most of the bacteria tended to aggregate and to be obvious around clumps of epithelial cells, and these cells were generally disregarded.

Results.

The inherent possibilities of genetic sex determination from the method of buccal smears are obvious in the photographs (Figures I, II and II), which are annotated. Gross bacterial contamination and folding over of the epithelial cells constitute difficulties, and differentiation is often impossible in plaques of epithelial cells.

There was close agreement in cell-counting by all three observers in a series of 25 "declared" normal males and in a similar number of normal females. Averages of closely agreeing individual counts by all three observers were: normal males, 5% female type cells; normal females, 61% female type cells. However, in our first small group of patients of indeterminate sex there were definite discrepancies in the results of cell differentiation recorded by the three observers. This is illustrated in Table I.

TABLE I.

Percentage of Female Type Cells Scored by Three Observers, R.R., J.E.Z. and G.R., in Four Patients of Indeterminate Sex.

Patient.	Percentage of Female Type Cells Recorded by			Average.
	R.R.	J.E.Z.	G.R.	
X1	53	59	16	39
X2	10	30	70	37
X3	54	17	67	45
X4	56	51	20	43

Discussion.

There was a variation in the opinions expressed by the observers about the four patients of indeterminate sex, although they expressed unanimity regarding the normal males and females. It would appear that the main points of dissension, after the problem had been discussed, rested largely on bacterial contamination and the introduction of artefact. This is unavoidable with mucosal smears, and could well have been the main factor for the diversity of opinions expressed; but it is difficult to reconcile this view with the more uniform results that were obtained in normal persons. Young (1937) has discussed the occurrence of gynandromorphism in moths and other insects and in a few birds. In this condition certain parts are genetically male and certain parts genetically female. Usually half of the body is male and half female, but very complicated mosaic formations have been known to occur. It has not been described in the human race, but there are chromatin mass changes in the cells in adrenal cortical hyperplasia and in hermaphroditismus verus, and these changes may be akin to those of the mosaic pattern of gynandromorphism. Each observer worked independently on two stained slides of cells from each patient; and it is more than likely that they examined many differing fields, which would also account for a difference in the results, especially in the indeterminate sex series.

Although this is not a clinical report, it may be mentioned briefly that three of the patients were babies aged under 11 months, and one was a child aged eight years. Each child had an enlarged clitoris, no palpable masses in the labia and varying degrees of vaginal formation, the vagina of one being a mere sinus. One of these babies has died recently from acute adrenal failure. The child aged eight years developed pubic hair at the age of 18 months. A laparotomy was performed at the age of two years and a biopsy was carried out. This revealed ovarian tissue on one side, but it is a pity that both gonads were not subjected to biopsy. After all, this child may well be a true hermaphrodite, as the untouched gonad may be a testis.

It is impossible to be dogmatic about the position which the buccal mucosal smear method should occupy in sex determination. It appears to be as reliable as skin and leucocyte drumstick methods (Nelson, 1956). The normal range adopted by Nelson is 4% to 5% female type cells for normal males and between 20% and 72% for normal females. There is a clear line of demarcation except in some doubtful cases.

As a preliminary investigation, it is suggested that the buccal mucosal smear method could provide a useful guide and possibly a definitive result. The cell count taken in conjunction with the serum 17-ketogenic steroid content should provide an answer to the necessity for laparotomy or other operative procedures.

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FUMIGATION WITH DICHLORETHYL ETHER AND CHLORDANE: HYSTERICAL SEQUELÆ.

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AND
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IN order to counteract the effects of a borer infestation, an experienced pest exterminator was engaged to spray the wooden foundation beams of a country clothing factory. A mixture of 12% dichlorethyl ether and 2% chlordane dissolved in kerosene was used. The first ingredient vaporizes quickly and exerts an immediate toxic action; the latter, due to its residual properties, minimizes the likelihood of re-infestation.

The factory was single-storied and had a floor area of approximately 18,000 square feet; individual manufacturing sections were separated by partitions eight feet high. The building was adequately ventilated by numerous windows, but, because of prevailing blizzardly conditions, they were kept closed whilst the spraying was in progress.

In order to reach the affected joists and wall plates the pest operator had to cut numerous openings, each approxi-

mately 12 inches by 17 inches, in the factory floor. Ground dampness made these boards swell and the "trap doors" could not be placed without prior planing of their edges. For this reason it would have been difficult for rising fumes to penetrate easily into the workrooms above.

Between June 21 and June 25, 1956, 60 gallons of the insecticide were hand sprayed, at pressures varying between 30 and 35 pounds per square inch, onto the 25,000 super feet of affected timbers. Because the floor joists were close together, it was necessary to use larger quantities of the mixture per square foot than is frequently the case.

As a result of difficulties in reaching some of the affected timbers, it was not possible, on successive nights, to spray areas of equal size. The highest quantity used per single session was 20 gallons. During the final spraying eight

level; fans were used in an attempt to "remove remaining fumes".

Because of Press and wireless publicity these unusual happenings quickly became common knowledge throughout the town. Within a period of 11 working days, 55 employees out of a total of 166 were affected to a greater or lesser degree. The distribution of this absenteeism may be seen in Table I and Figure I. Those affected were away from work for a total of 218 days.

On July 6, as a result of requests made by the factory manager and pest exterminator, a doctor and a scientific officer of the Division of Industrial Hygiene visited the factory to determine the cause and nature of this "epidemic". During the five hours their investigations lasted, neither was adversely affected.

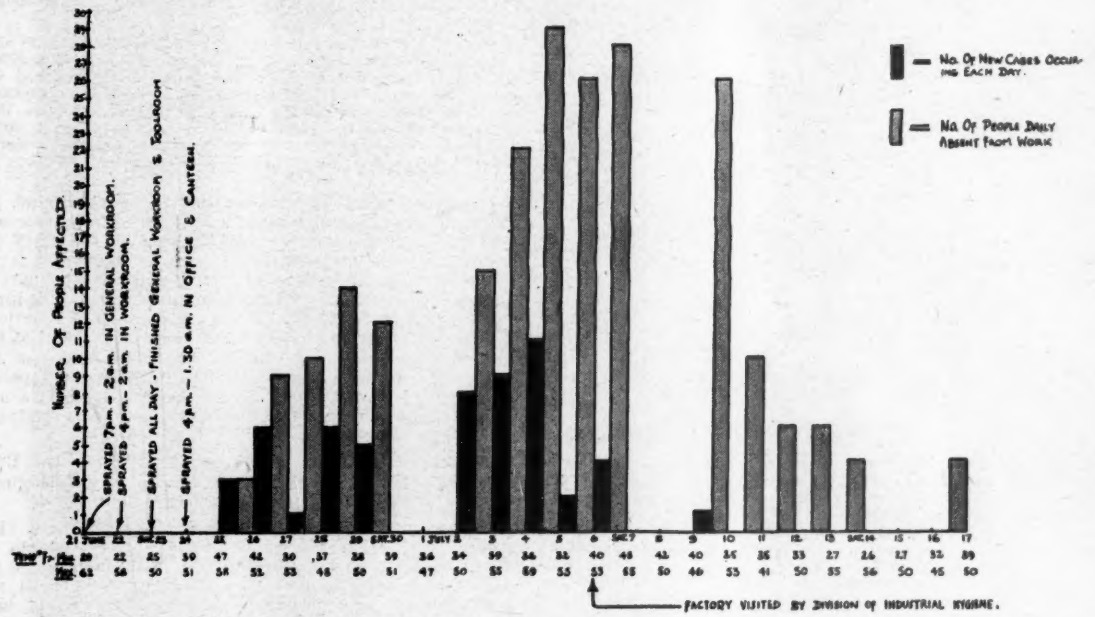

FIGURE I.
The distribution of absenteeism.

factory employees were working overtime until 10 p.m.; one was affected several days later. At no time was the pest control operator ill.

About midday of June 25, a young girl, aged 19 years, working in the marking room, complained of a mild skin irritation of her chest and face. Later she fainted and, on recovery, was sent home by taxi. Within a few hours two of her workmates were also affected. At this stage nobody was unduly alarmed. The next day, about noon, another girl fainted. The ambulance was summoned and after the driver had been inside the factory a little while he complained of a strong smell and then collapsed. Management was now worried and recalled the sprayer for his advice. He arrived in time to see two more girls collapse: "Their legs folded up from under them and the girls gently subsided to the floor."

Within a short space of time 12 more employees were affected. Others developed a slight rash on their exposed skin; this was likened to a "flush" which, in many instances, quickly disappeared in the open air. All employees were allowed to leave the local hospital within an hour or two.

At this stage management decided to close the factory temporarily. All doors and windows were opened wide and multiple holes were made in the brick walls below floor

Investigations Made.

Scientific.

In order to exclude the possibility of carbon monoxide poisoning (from the 40 suspended gas radiators used to heat the factory) several tests, using a Siebe Gorman potassium pallado-sulphite detector, were made during the course of the day. While the heaters were lit windows and doors were purposely kept closed.

The factory atmosphere was also sampled at multiple points for the presence of organic chemical compounds. The apparatus used consisted of an absorption tower connected to a silica tube, containing a platinum catalyst, and heated to 1000° C. in an electric furnace. The air sampled was moistened by passing it over wet cotton wool. In the hot part of the tube any chlorinated hydrocarbons present would be converted to water vapour, carbon dioxide and hydrochloric acid, the latter being absorbed in the saturated sodium carbonate solution contained in the tower. After titration with silver nitrate, it was possible to relate back the acid to the molecular weight of the chlorinated hydrocarbon sampled.

Both series of tests gave negative results, and it was clear that there was no toxic agent persisting in the factory atmosphere. It was, however, possible to detect a slight odour of kerosene.

Medical.

As it was not possible to see every employee, nine of the girls and women currently affected were interviewed and examined. Their ages varied from 16 to 52 years. Six worked in the marking section, two in the machine room and one in the overlock department. In Table II the main symptoms and signs for eight of these people are tabulated. The ninth girl interviewed, aged 16 years, was one of the first adversely affected. Because she "took a characteristic turn" while the medical officer was present, it is proposed to record fuller details.

This employee felt well until morning tea time on the second day after the spraying had finished. She then "noticed a peculiar smell and just went out to it". According to eye witnesses, she was "unconscious but fought everyone". Sore eyes, headache and cramps in the stomach subsequently developed, together with multiple "mottled red patches" on the face and neck. There was no irritation of the respiratory tract. Forty-eight hours later she had to leave work because she was flushed and felt sick. She recovered within four days and her doctor told her that "there was nothing wrong". However, on returning to work she soon felt sick again and complained of "stinging of the eyes". On July 5 her face and neck were flushed; she also had a "sore throat and couldn't talk or breathe properly"; while sitting on an outside step she went "into a sleep and tried to fight everybody again; it took a lot of people to hold her down". Eye-witness accounts do not suggest a fit; there was neither biting of the tongue nor passage of urine or fæces. There were no convulsive movements.

TABLE I.
The Distribution of Absenteeism.

Absenteeism. (Working Days.)	Number of People.
Up to one-half ..	9
One	12
Two	1
Three	5
Four	5
Five	6
Six	9
Seven	2
Eight	1
Eight to ten	1
Ten to twelve ..	3
Twelve or more ..	1
Total ..	55

On the day this girl was examined by a medical officer of this division, she complained of a sore throat and sore eyes in addition to frequent alternate hot and cold shivers. She felt as though she was "developing a cold in the head" and at times complained that it was "hard to breathe". On examination, the pulse and respiratory rates were respectively 92 and 28 per minute; both were regular. During her medical examination she complained of feeling sleepy and wanted to lie down. Fellow workers commented that "she must not be allowed to go to sleep because she would take one of her turns again". About half an hour later the doctor was called into the locker room, where this girl was lying slumped on the floor. The respiratory rate was now 40 per minute and stertorous; her tongue was causing a partial obstruction which disappeared when she was straightened up. The pulse rate was regular and 130 per minute. Both pupils were dilated and reacted to light. Knee and ankle jerks were present though exaggerated on both sides. A chest examination revealed no abnormality. There was no skin rash. Although this girl was conscious and able to answer questions while being examined, she made no protest when firm pressure was applied over the supraorbital nerves.

After all employees had returned to work we attempted to ascertain the stated reasons for their absenteeism. The analysis is shown in Table III.

Physical Properties and Toxicology of Chemicals Used.

Chlordane.

Chlordane is dark amber in colour. It is a highly viscous liquid with a slight terpene-like odour. The total chlorine content is 69%, and its volatility lies between that of DDT and BHC. "Technical chlordane" contains between 60% and 75% of 1,2,4,5,6,7,8,8,octachloro-4-7-methano 3a,4,7,7a tetrahydroindane; the remainder consists of related dicyclo pentadiene derivatives. The varied uses of chlordane, together with its effect on animals and plants, have been recorded in detail (Roark, 1951). The fatal dose for man has been variously estimated at between five and 60 grammes (Lehman, 1949). Clinical details of numerous accidental or intentional swallowings have been described in the literature (Lensky and Evans, 1952; Derbes *et alii*, 1955). We wish to draw the reader's attention to the following examples of acute ill-effects.

A young housewife slept in an unventilated room which, several hours previously, had been sprayed with a 2% solution of chlordane. Several days later she was admitted to hospital with a mild upper respiratory tract infection and hepatic dysfunction. The patient was a heavy drinker (Lemmon and Pierce, 1952).

A ten-months-old child was accidentally sprayed about the head and neck with a 2% solution of commercial chlordane and, in spite of being immediately washed with soap and water, within an hour became hyperirritable and cried hysterically for at least 30 minutes (Micks, 1954). It is of interest to recall that in poisoning with aldrin, another chlorinated hydrocarbon insecticide, electroencephalographic changes have been recorded (Spiotta and Winfield, 1952).

From the point of view of dematitis, chlordane, particularly the undiluted product, can be moderately irritating. This was especially the case in the early days of its manufacture. When dermatitis occurred it was frequently difficult to exclude with certainty the part the kerosene vehicle played in the chain of events (Goldman, 1950). "In 1951 the impurities which had no particular insecticidal activity were pruned down and removed from the technical product. Since then, chlordane has been recognized as being less hazardous to humans, especially in regard to skin contact" (Ingle, 1956). Recently a death occurred as a result of extensive skin contamination (Derbes *et alii*, 1955).

The threshold limit established in 1956 by the United States Conference of Governmental Industrial Hygienists was 2·0 milligrammes per cubic metre of air.

It is of interest to note that Princi and Spurbeck (1951) studied 22 workers exposed to chlordane, aldrin and dieldrin fumes during the course of manufacture; in spite of occasional atmospheric concentrations up to 54 milligrammes of chlorinated hydrocarbons per cubic metre, abnormalities were not detected during subsequent clinical and laboratory investigations.

Dichlorethyl Ether.

Dichlorethyl ether (DICE) is more volatile than chlordane. At a concentration of 35 parts per million of air it is possible to detect its odour (Brown, 1951). The threshold limit has been set at 15 parts per million of air. Depending upon the amounts inhaled, a mild respiratory tract irritation or chemical pneumonitis may develop. The latter may also be caused by long exposures to low concentrations not causing any immediate response.

> The establishment of tolerances for mixtures has been under consideration . . . for several years, but because of the complexity of the problem no workable approach has as yet been found (Bale, 1956).

Discussion.

Apart from using slightly larger quantities of the insecticides, the methods used at the factory did not differ from the common practice of most other operators when fumigating private dwellings. Moreover, on many occasions the top surfaces of floors of homes have been sprayed early in the morning and the owner has slept, without ill-effects, in the same room at night; however, a few occupants have complained of transient discomfort from the smell of dichlorethyl ether and/or kerosene.

In order to gain additional information on resulting vapour concentrations, we recently performed further investigations after a timber home had been sprayed. On

TABLE IIA.

Patient.	Monday (June 25, 1956).					Tuesday (June 26, 1956).			
	Detected Odour.	Headache.	Sore Eyes.	Skin.	Miscellaneous.	Headache.	Sore Eyes.	Skin.	Miscellaneous.
1	Yes.	Yes.	Yes.	—	—	—	—	—	—
2	Yes.	Yes.	—	—	Sore tongue and painful jaw.	—	—	—	—
3	—	Yes.	—	—	—	—	—	—	—
4	—	—	Yes.	Transient erythema of anterior chest; face also itchy.	Felt faintish.	—	—	—	—
5	Yes.	—	—	—	—	Yes.	Yes.	Face and neck flushed.	At times felt "hot and cold". Vague abdominal pains present. Cried easily.
6	—	—	—	—	Fainted after she saw a girl develop a rash.	—	—	—	—
7	—	Yes.	—	—	—	Yes.	Yes.	"Prickly sensation all over."	Fainted after seeing other girls doing likewise. Felt alternatively hot and cold, and as if she "had a weight" on her chest.
8	—	—	Yes.	Erythema "all over", especially throat and upper chest.	—	—	—	Rash all over.	"I carried on a treat; I got hysterical."

TABLE IIB.

Patient.	From June 27, 1956, to July 5, 1956.					July 6, 1956 (Date of Examination by Divisional Medical Officer).					
	Headache.	Sore Eyes.	Sore Throat.	Skin.	Miscellaneous.	Headache.	Sore Throat.	"Shivery Attacks."	Miscellaneous.	Medical Findings.	Details of Absenteeism.
1	Yes.	—	—	Face repeatedly flushed.	Four days after spraying she felt weak; repeated attacks of alternatively hot and cold. Tendency to faint and cry at least provocation.	Yes.	Yes.	Yes.	Had had a sore neck.	Nil.	Three-quarters of July 6.
2	—	—	—	—	Nil.	Yes.	—	Yes.	Occasional palpitations.	Nil.	Half of July 4.
3	—	—	—	—	"Pins and needles" in arms. Pains over heart. Previous history of hypertension.	—	—	—	—	Conjunctivitis, mild herpes labialis.	Nil.
4	—	—	—	On repeated occasions she developed a flushing of face, neck and chest.	Nil.	—	—	—	—	Slight erythema round sternum.	Half of June 26, three-quarters of June 27, whole of June 28 and July 4 and 6.
5	Yes.	Yes.	—	Rash on face and neck developed which was "always present till out of the factory".	Nil.	Yes.	Yes.	—	Felt sick after morning tea.	Nil.	June 26 to July 2.
6	Yes.	Yes.	Yes.	—	Backache. "Felt as though could easily faint. Wanted to cry all the time."	Yes.	Yes.	—	Dizzy all day; "felt as though could sit down and scream". Breathing difficult at times.	Flushed face and neck; conjunctivitis.	Half of June 26, three-quarters of June 27, whole of June 28.
7	—	—	—	—	Nil.	Yes.	—	Yes.	Could not concentrate; skin felt prickly.	Slight redness of the left arm and forearm.	June 26 to July 9.
8	—	—	—	Three days after spraying developed a "rash all over".	—	Yes.	—	—	"Felt like fainting with the smell"; "rash goes away when I settle down".	Flushed face.	Half of June 25, 26 and 28, all of June 27.

this occasion six gallons of a 2% chlordane, 20% dichlorethyl ether solution in kerosene were applied to the underside of the hardboard floor boards (1000 square feet); the top surfaces were bare. Spraying started at 9 a.m. and was completed some three and a half hours later. Our first test was made at 3.30 p.m. On entering the house, the insecticide could be easily smelt; in addition, one of us noticed a slight transient nasal and eye irritation. Twenty-seven hours after completion of spraying, a second air analysis was made; by then the odour was extremely faint. At no time did either the housewife or her small son complain of undue irritation. Total chlorides were calculated for both samples; on the basis of comparative vapour pressures we assumed that any chloride detected would be due to the DICE.

Table III.

Sickness Analysis.

Illness Stated on Medical Certificate.	Number of People.	Illness of Patients Who did not Attend a Medical Practitioner.	Number of People.
Chemical dermatitis ..	5	Skin irritation	18
Dermatitis and faintness	2		
Dermatitis and headache	1		
Dermatitis, headache and giddiness ..	2		
"Toxic effect" from fumes	11	"Toxic effects" from fumes	6
Upper respiratory tract irritation	4		
Debility from fumes ..	2		
Headache and backache	2		
Headache, backache and pyrexia	1		
Giddiness and nausea	1		
Total	31	.. ..	24

Results.

Three hours after spraying, 2·5 parts per million of DICE were detected; 27 hours after spraying, no DICE was detected. In view of the latter figure no further tests were made.

It is conceivable that during the first day or two after cessation of spraying there may have been sufficient DICE and kerosene vapours in the general atmosphere of the different factory workrooms to cause a mild irritation of the eyes, nose and throat. Initial concentrations would not have been sufficient to cause loss of consciousness.

We calculate that at the end of the fourth day of spraying a maximum concentration of 750 parts per million of air of dichlorethyl ether, 40 parts per million of air of chlordane and 3200 parts per million of air of kerosene vapour would have been present if (i) the factory had been completely sealed throughout the whole period, and (ii) all of the mixture had penetrated through the floor boards and quickly dispersed throughout the general factory atmosphere. Concentrations must have been considerably less than those quoted above because of (*a*) the slow rate of evaporation, (*b*) the tight floor boards, and (*c*) the general ventilation of the factory.

Calculations suggest that the maximum possible concentrations likely to have been experienced by employees seven hours after cessation of spraying was in the vicinity of one to two parts of DICE, 0·06 part of chlordane, and 15 parts of kerosene per million of air. It is possible that, as a result of using the gas heaters, slightly higher concentrations may have been present at least for a few hours. It is difficult to arrive at a precise figure.

Because there would be little or no absorption of vapours, we consider that neither the handling of the textiles nor the wearing of clothing exposed to the contaminated atmosphere would cause trouble. Neither had been deliberately sprayed. Moreover, in many cases the affected regions were not clothing "contact" areas.

It is possible to subdivide the medical findings into two broad groups.

Signs and Symptoms of Initial Local Irritation.

The eyes and skin were chiefly affected. Some of the transient flushings "were the mottled irregular vasodilation frequently seen in the surgery on nervous women" (comment of Dr. Docker). Other employees were developing, or had just had, the respiratory infection currently prevalent in the community.

Anxiety, Fright or Hysteria.

It is not clear why a state of mass anxiety developed. Many employees complained of "palpitations", "pins and needles", "hot and cold shivers", "weeping dizziness", "the shakes" or "a weak feeling all over". In addition, others behaved hysterically; to quote "I saw all the others fainting and I just fainted too", "I wanted to sit down and scream", "I got hysterical and carried on a treat", or "I cried myself to sleep".

The five doctors who attended the affected employees were interviewed; four agreed that many of the symptoms could be explained on a basis of mass anxiety or group hysteria; two of these practitioners also considered that there had been an initial irritation. The fifth preferred not to pass any comment.

On July 11, 1956, a letter giving the results of our investigations was sent to the manager with the request that the employees be informed of its contents. Our report concluded:

> It is our considered opinion that:
>
> 1. There is now no contamination of the atmosphere of the factory with any poisonous or irritating substances.
>
> 2. The symptoms and signs presented by those questioned and examined are not consistent with the known effects of the insecticides used.
>
> 3. Although initially some irritation may have been present, this should have disappeared within a short period. Instead, for some reason there has developed a mass anxiety phenomenon amongst the employees which is accounting for the present complaints.
>
> If the above facts are made known to the employees, and the necessary reassurances given, there should be no continuation of present troubles.

After this report was received no new cases occurred, although one employee, who had already been away several half days, was away from work the whole of the following week.

> Nothing in life is more remarkable than the unnecessary anxiety which we endure and generally occasion ourselves.—Disraeli, statesman, novelist and poet, 1804 to 1881.

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Finally we wish to thank the Director General of Public Health, New South Wales, for permission to publish this article.

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Reviews.

The Lipids: Their Chemistry and Biochemistry. Volume III. By Harry J. Denel, jnr.; 1957. New York, London: Interscience Publishers, Incorporated. 9" x 5¾", pp. 1102, with illustrations. Price: $25.00.

THIS is the third and last volume of the monumental work of Harry J. Denel, junior, on lipids. It was also the last work of Dr. Denel before his untimely death. This volume deals in great detail with the biosynthesis and breakdown of triglycerides, phospholipids, steroids, fat-soluble vitamins and other commonly occurring compounds. In addition, it has a chapter on the metabolism of branched chained acids, hydroxy and keto acids, hydrocarbons and other less common compounds. This chapter is particularly valuable, as much of the material is hard to find in other reviews and books. In addition to metabolism, the nutritional value of the various compounds is also discussed. It is difficult for a single author covering such a large field to make a critical evaluation of all aspects discussed. Some chapters, such as those dealing with the metabolism of fatty acids and phospholipids, where recent research has been particularly intensive, are out of date, and for these topics better and more critical reviews are available. However, in spite of these shortcomings, the book is an invaluable reference work and should be in all libraries catering for readers interested in biochemical topics.

Physics for the Anæsthetist: Including a Section on Explosions. Edited by Sir Robert Macintosh, D.M., F.R.C.S.E., F.F.A.R.C.S., M.D., W. W. Mushin, M.A., M.B., B.S., F.F.A.R.C.S., and H. G. Epstein, M.A., Ph.D., F.F.A.R.C.S.; Second Edition; 1958. Oxford: Blackwell Scientific Publications. 8¾" x 5½", pp. 455, with many illustrations. Price: 60s. (English).

ANÆSTHETISTS, particularly candidates for higher degrees, will welcome the new edition of this valuable book. It must be five years since this book has been available for sale in this country. During this time owners of the first edition, or its only reprint in 1947, have been guarding their volumes as prized possessions and almost as collector's pieces. It is pleasing to note that the physicist in the Department of Anæsthesia at Oxford, H. G. Epstein, has now been named as a co-author. The original authors, R. R. Macintosh and W. W. Mushin, acknowledged his valuable aid in the preface to the first edition.

The size of the book has almost doubled in volume, but the format remains the same. The increase is due to the elaboration of certain paragraphs, as where the description of reducing valves is increased from one paragraph to one chapter, and particularly to the addition of four chapters on oxidation, combustion, deflagration and detonations in fuel mixtures. With the usual profuse use of black and white diagrams, and some in colour, these interesting chapters add the new bulk to the book. Physical data and biographical notes have been revised, slightly enlarged and retained at the end of the descriptive section.

It is difficult, almost impossible, to be critical either of the contents or the printing of the book. The text is easy to read and, as in the first edition, profusely illustrated with simple explanatory diagrams and graphs conveniently placed relative to the text. Perhaps it is disappointing to find that specification for anæsthetic apparatus has not been included, either in the body of the work or in the section on physical data, especially as the authors have taken such trouble, and rightly so, to point out the dangers and disadvantages of restrictive apparatus. This matter has now been under consideration by the National Standards Association for some time, and a printed code should soon be available.

It is interesting to read in the preface to this, the second edition, the reason for the original publication of this work. While teaching anæsthesia, the authors found that, to answer questions of post-graduate students, reference had to be made to books on physics and medical physics as existed then. Neither reference supplied them with a simple answer, and "scientific accuracy had to be reconciled with brevity". Unlike many other text-books in medicine, which are a reiteration of many other published works on the same subject, this book is unique in that there is no other publication like it. Therefore, it fills an important gap in our reading. There being no other such book available with a complete review of physical laws applicable to the practice of anæsthesia, no comparison can be made. Even if there were, it would be difficult to imagine such a publication approaching the excellence of this work in its clarity, content and presentation.

The book is, of course, essential for the specialist anæsthetist and for candidates approaching their specialty, and the chapters on detonations and deflagrations should be read and remembered by all medical practitioners handling inflammable material.

General Diagnosis and Therapy of Skin Diseases: An Introduction to Dermatology for Students and Physicians. By Hermann Werner Siemens, M.D.; 1958. Chicago: The University of Chicago Press. 9¼" x 6¼", pp. 236, with 375 illustrations. Price: $10.00.

THIS introduction to dermatology for students and physicians has been admirably translated from the German edition by Kurt Wiener (Milwaukee, Wisconsin). The book aims to teach dermatology by thorough explanation of fundamentals and by the use of 275 black and white close-up photographs of the highest quality. These photographs have been taken under the direction of the author at his skin clinic at the University of Leiden, Holland. They adjoin the relevant parts of the text, making ready reference possible.

The fundamental rules of treatment are well laid down, and the student is encouraged when treating his patients to use those measures that have been proven by experience rather than to experiment indiscriminately. The author warns against blind adherence to traditional methods of treatment and stresses the need for the student to acquire and maintain a spirit of scientific curiosity. However, the early section on diagnosis is too detailed, and the description of fundamental lesions, although precise, is based too slavishly on morphology. It is not necessary or desirable to give a name to every minute detail of observation. By so doing the student clutters up his mind with a profusion of descriptive terms denoting changes in the skin, where a good knowledge of physiology and histology would lead to greater understanding. For the same reason, the general practitioner would need to study this book intensively for a long time even to fix the names in his mind. Such detail creates confusion. Similarly, the specialist dermatologist does not need to memorize the names of such changes. It is time that dermatologists made an earnest endeavour to steer away from the old traditional methods of description and to simplify terminology, particularly that relating to ætiology. This book does not help much in this regard.

In general, the book cannot be regarded as adding anything new to dermatology as the author intended, although it does add to the number of good photographs available for study.

Tumors of the Soft Somatic Tissues: A Clinical Treatise. By George T. Pack, M.D., LL.D., F.A.C.S., and Irving H. Ariel, M.D., F.A.C.S.; 1958. New York: A Hoeber-Harper Book. 10" x 6¼", pp. 804, with 652 illustrations. Price: $30.00.

As a study of a heterogeneous collection of conditions, arising in soft tissues, this is a formidable work. It is clinical in its approach and gives the results of an extensive experience. It also gives a limited but reasonably valuable review of the literature.

It is by no means scientific in outlook. The definition of the subject matter is of "mesodermal tissues", but tumours of tissues which, though "soft", are not mesodermal are included. In view of the title, perhaps one cannot reasonably criticize the material included, and the catalogue includes not only connective and muscle tumours, but also

certain growths of the kidney, breast, carotid body and other structures. The inclusion of several conditions such as Dupuytren's contracture, keloid and congenital torticollis is an indication of the catholic interests, if not the scientific appreciation, of the authors.

The description of procedures is good and well illustrated, and ranges from simple removal of localized tumours to hindquarter amputations and hemipelvectomies. However, the student must look elsewhere for some of the technical details of most procedures. It is in this aspect and in the results obtained by the writers that the main purpose of the book is found. Incidentally, it should be noted that percentages often have little meaning in small groups of cases.

As has been indicated above, the discussion of the pathology of most of the conditions is superficial and even, in places, out of date or misleading. For this reason the presentation could not be recommended to the student who has not progressed sufficiently far to have developed a critical (and indeed sceptical) attitude of mind. It should be said, nevertheless, that much of the superficialities and inadequacies of the approach to the problems inherent in the conditions reviewed may well arise from the necessity of attempting to compress so much into the relatively small space of this work.

There is a certain amount of carelessness in the final checking of the text, in that some parts are repeated word for word in different sections. This seems an unnecessary extravagance and demand on the patience of readers which is unwarranted in a book of this size. It weighs six pounds, though it is doubtful if this will be regarded as recompense for the price asked for it. Nevertheless, a large amount of material is presented in an attractive form, and the book may well be found of interest and value to some busy clinicians.

Antisera, Toxoids, Vaccines and Tuberculins in Prophylaxis and Treatment. By H. J. Parish, M.D., F.R.C.P.E., D.P.H.; Fourth Edition; 1958. Edinburgh and London: E. and S. Livingstone, Limited. 8½" × 5", pp. 266, with many illustrations. Price: 30s. (English).

THIS trusted standby of the medical practitioner has appeared in its fourth edition within ten years. More biological products are listed, and there are more plates, both in black and white and in colour, but essentials have not changed. The style is simple, and discussion is straightforward and restricted to points of immediate interest in medical practice. Undesirable side-effects and inherent dangers are discussed for every product.

The first chapters give an outline of general problems and of the basic concepts of immunology. The uses of prophylactics, antisera and diagnostic biologicals are described and criticized. A few paragraphs are devoted to the sterilization of syringes, but reference to sterilization by boiling and hot-oil sterilization could have been omitted or at least clearly designated as emergency measures; the use of the pressure cooker when more elaborate apparatus is unavailable could be recommended instead. The main divisions of the book contain information on antisera, products for active immunization and diagnostic biologicals. Each chapter treats of one product, such as antivenenes, bacterial vaccines, poliomyelitis vaccine or tuberculins. Facts are presented, and a short discussion follows. Frequent references are made to official publications of the British Ministry of Health. The book ends with a concise historical review (an error or misprint gives 1933 as the date of publication of Weil-Felix's diagnostic agglutination in typhus fever) and a list of references to text-books and monographs.

The Facts of Mental Health and Illness. By K. R. Stallworthy, M.B., Ch.B.; 1958. New Zealand: N. M. Peryer, Limited. 8¼" × 5", pp. 218. Price: 22s. 6d. (New Zealand).

K. R. STALLWORTHY is Senior Medical Officer, Auckland Mental Hospital, Auckland, New Zealand. For three years he lectured on mental hygiene to adult education classes. At the request of his students he made a written record of his lectures. This is the second edition of his work, which is an enlargement of the first edition. In the preface to his work, Dr. Stallworthy says that an unenlightened dread of mental hospitals is less common than it used to be, but is still an important factor in adding unnecessarily to the heart-break which mental illness may bring, and in hindering recruitment to under-staffed mental hospitals; for that reason only he stresses the facts about mental hospitals, what they do and what they are. His book is written for the layman, and he covers the whole field of his subject so ably that there should not be any hesitation in recommending it to anyone who, it is considered, would be likely to benefit from a knowledge of its contents. Dr. Stallworthy's advice on the bringing up of children is helpful, as also are his suggestions for moulding our own conduct in the interests not only of our own mental health but also of that of those persons with whom we are in frequent contact. The book is most interesting and informative throughout.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The Brain and Human Behavior"; 1958. Baltimore: The Williams and Wilkins Company. 9" × 5¾", pp. 575, with 200 illustrations and 53 tables. Price: £8 5s.

Proceedings of the Association for Research in Mental and Nervous Disease, December 7 and 8, 1956, New York, N.Y.

"Alcoholism", by Arnold Z. Pfeffer, M.D.; 1958. New York, London: Grune and Stratton. 8½" × 5", pp. 112. Price: $6.50.

One of the "Modern Monographs in Industrial Medicine" series.

"The Art of Clinical Refraction", by Theodore H. Whittington, M.D., M.R.C.P., D.O.M.S.; 1958. London: Oxford University Press. 8½" × 5½", pp. 300, with illustrations. Price: 54s. 6d.

Based on lectures and practical teaching given to postgraduates at the Royal Eye Hospital and to students at King's College Hospital.

"Practical Cardiology", by Albert Salisbury Hyman, M.D., F.A.C.P., F.A.C.C.; 1958. New York: Landsberger (Medical Books), Inc. 8" × 5", pp. 322. Price: $7.00.

This is one of the "Handbooks for the General Practitioner" series.

"Anomalies of Infants and Children", by D. McCullagh Mayer, D.D.S., M.D., F.A.C.S., F.I.C.S., and Wilson A. Swanker, M.D., F.A.C.S., F.I.C.S.; 1958. New York, London: McGraw-Hill Book Company, Inc. 8½" × 5½", pp. 464, with 95 illustrations. Price: $12.00.

"This book is an attempt to place in a single volume our present knowledge of the more common types of congenital and acquired abnormalities."

"Leptospirosis in Man and Animals", by J. M. Alston, M.D., F.R.C.P., and J. C. Broom, O.B.E., M.D.; 1958. Edinburgh and London: E. and S. Livingstone, Limited. 8½" × 5", pp. 380, with many illustrations. Price: 40s. (English).

The authors' aim has been to tell what is known about leptospirosis and to show where new knowledge is at present being gained and may be hoped for in the future.

"Cardiovascular Diseases", by David Scherf, M.D., F.A.C.P., and Linn J. Boyd, M.D., F.A.C.P.; Third Edition; 1958. New York and London: Grune and Stratton. 9½" × 6½", pp. 846, with many illustrations. Price: $17.75.

A revised and enlarged edition.

"Sir Charles Bell: His Life and Times", by Sir Gordon Gordon-Taylor, K.B.E., C.B., F.R.C.S., and E. W. Walls, M.D., Ch.B., B.Sc., F.R.S. (Ed.); 1958. Edinburgh and London: E. and S. Livingstone, Limited. 8½" × 6", pp. 300, with illustrations. Price: 42s. (English).

The story of a great doctor set against the background of notable times.

"The Proceedings of the Medico-Legal Society of Victoria: During the Years 1955 to 1956", edited by G. H. Lush, LL.B., and Kenneth J. Grice, M.D., M.R.C.P., F.R.A.C.P.; Volume VII; 1957. Melbourne: Brown, Prior, Anderson, Proprietary, Limited. 8½" × 5", pp. 171. Price not stated.

Contains seven papers read before the Society and an after-dinner speech by Professor Maurice Ewing.

"Diseases of the Thyroid and Parathyroid Glands", by Bernard J. Ficarra, A.B., Sc.B., M.D., D.S.; 1958. New York: Intercontinental Medical Book Corporation. 9" × 5¾", pp. 310, with 130 illustrations. Price: $8.50.

A comprehensive treatment of the subject based on extensive experience. The author is a surgeon.

The Medical Journal of Australia

SATURDAY, AUGUST 23, 1958.

THE INTERIM REPORT OF THE NATIONAL RADIATION ADVISORY COMMITTEE.

THE importance to the medical profession of a knowledge of radiation's uses and its hazards is obvious, and it is the duty of doctors to keep themselves informed on the subject. Few among us would claim the knowledge of, for example, physics required to grasp many fundamental aspects of the subject, but we do have available enough objective and not unduly technical information to keep us in the picture. If some of it is conflicting, that is nothing new to those accustomed to reading medical literature. There is enough that is beyond reasonable question to keep us thoughtful. Of particular interest in this respect is the interim report made to the Prime Minister in July of this year by the National Radiation Advisory Committee. This Committee was appointed by the Commonwealth Government in May, 1957, to advise the Government, directly through the Prime Minister, on any matter concerning the effects of ionizing radiation on the Australian community, whether arising from medical, industrial, scientific, international or other causes. The Committee consists of the following: Sir Macfarlane Burnet, as chairman; Professor S. Sunderland, Professor of Anatomy in the University of Melbourne; Professor Sir Leslie Martin, Professor of Physics in the University of Melbourne; Professor J. P. Baxter, Chairman of the Australian Atomic Energy Commission; Professor E. W. Titterton, Professor of Nuclear Physics in the Australian National University; Mr. D. A. Gill, Chief of the Division of Animal Health and Production of the C.S.I.R.O.; Dr. W. P. Holman, Medical Director of the Cancer Institute Board of Victoria; Mr. D. J. Stevens, Director of the Commonwealth X-Ray and Radium Laboratory; Mr. J. R. Moroney, of the Department of Supply, who is the Secretary of the Committee. Mr. E. L. Cook, Assistant Secretary of the Research and Development Branch of the Department of Supply, has also attended all meetings at the invitation of the Chairman. The present interim report has been presented because the Committee considers that the time is opportune to do so, a reason provided for in its terms of reference.

The report, which is commendably brief yet informative, presents first of all summaries of current knowledge of the biological effects of ionizing radiation and of the sources of ionizing radiation to which man is exposed. Then follow an account of various matters considered by the Committee, a summary of the Committee's recommendations and some suggestions for further reading. Three matters of direct medical interest have been considered by the Committee: the use of X rays in medical diagnosis, tuberculosis case-finding programmes involving the use of mass radiography, and radiation-induced leukæmia. The Committee recognizes that the medical necessity for a particular X-ray diagnostic procedure can be evaluated only when all clinical implications are taken into account by the medical practitioner. However, it takes the view that the use of X rays in medical diagnosis is likely to remain the major contributor to man's radiation exposure if the great advantages the practice provides to his health and well-being are not to be sacrificed. It acknowledges that the medical profession individually and collectively is increasingly conscious of the problem and is seeking a solution. Nevertheless, apart from the specific recommendations made by the Committee to which we shall refer later, the report provides an added stimulus to the medical profession in its consideration of these matters. The report then refers to the various hazards which are or could be associated with mass radiography, but expresses the opinion that the risk is very small indeed and should be accepted at the present time. The practical value of mass X-ray chest surveys is fully recognized. Turning to the question of the rise in incidence of leukæmia, the report makes clear just how little is known about this and its causes, and it acknowledges the probability that ionizing radiation is at most only one of the factors concerned. The Committee's view apparently is that the only constructive approach to the subject of leukæmia at the moment is to find out more about it, and it makes its recommendations accordingly. In passing, mention may be made of the other matters considered by the Committee. One relates to the radioactive fall-out associated with atomic weapon tests in Australia. The Committee appears to be in no way alarmed by this, and reports that the most stringent safety precautions have been applied throughout. On the question of global fall-out, it is reported that the fall-out in Australia is extremely low, and certainly lower than that in most other countries. An evaluation of the situation in regard to strontium 90 is to be made available later.

The following is a summary of the specific recommendations made by the Committee:

> (i) Action similar to that taken by certain States in using the Model Act and Regulations prepared by the National Health and Medical Research Council to bring many of the sources of ionizing radiation under legislative control should be taken by the other States as soon as possible.
>
> (ii) Radiation doses to the individual and to the population as a whole arising from the medical use of X rays for diagnostic purposes should be reduced without delay by administrative and technical action.
>
> (iii) The strongest consideration should be given to the licensing of X-ray equipment to be used by medical practitioners for diagnostic purposes.
>
> (iv) The need for carrying out mass X-ray chest surveys in the programme to reduce the incidence of tuberculosis should be kept under review and a re-evaluation made from time to time of the advantages of this procedure in relation to public health.
>
> (v) All types of leukæmia should be declared notifiable diseases and a consultative panel of pathologists should be set up in each of the major population centres to establish diagnosis in difficult and borderline cases.

There will be mixed reactions to some of these recommendations, but in the main they must be supported. The Model Act and Regulations were prepared with great care by the National Health and Medical Research Council and have already been the subject of careful scrutiny.

Individual State governments have had enough time to decide whether they are prepared to reject them or implement them without further delay. Some may see in the recommendations regarding the medical use of X rays and the licensing of X-ray equipment the possibility of bureaucratic interference with medical practice. In general terms such a possibility is usually something to be deplored and perhaps resisted, but we doubt if this is the sort of thing the Committee has in mind. If the medical profession accepts these recommendations as a challenge to keep its own house in order rather than as an occasion to get up in arms, it should have little to fear. The recommendation with regard to mass X-ray chest surveys is an expression of the common-sense view with which no one is likely to quarrel. The recommendation about declaring leukæmia a notifiable disease raises an old issue—that of whether it is right to make notifiable any diseases other than the infectious group, notification of which is required for the protection of the whole community. This will need careful thought, as there is a principle involved. The Committee's motives in making the recommendation are not to be questioned for one moment, and everyone will acknowledge how valuable it would be to have this information. Perhaps it would be possible to devise some system of voluntary notification involving the patient's consent.

CHILDREN'S MEDICAL RESEARCH FOUNDATION.

On the day of publication of this issue of the Journal (Saturday, August 23, 1958) the public appeal is to be launched for funds to establish a Children's Medical Research Foundation in Sydney. An unusual and, for Australia, quite new feature of the launching will be a 27-hour continuous appeal on behalf of the Foundation over television station ATN (Channel 7). On this same day *The Sydney Morning Herald* will open its columns to acknowledge all individual donations. The ultimate objective of the appeal is the raising of £500,000 for the establishment and maintenance of a Children's Medical Research Foundation within the precincts of the Royal Alexandra Hospital for Children, Camperdown. The Patron is His Excellency the Governor of New South Wales, Lieutenant-General Sir Eric Woodward, the Vice-Patron is Sir Charles Bickerton Blackburn, the President is Sir John Northcott and the Chairman of the Executive Committee is Professor Lorimer Dods.

A citizens' meeting convened by Sir John Northcott and presided over by the Right Honourable the Lord Mayor of Sydney, in the presence of His Excellency the Governor of New South Wales, was held in Sydney on June 19 of this year. That meeting unanimously supported the establishment of a Children's Medical Research Foundation, and we may be confident that those present spoke for the majority of their fellow citizens. In his endorsement of the appeal for the Foundation, Sir Charles Bickerton Blackburn, Chancellor of the University of Sydney, has stated:

> Many millions are today being spent upon trying to prevent so many of those who reach the fifth and sixth decades from prematurely losing their lives from cardiovascular disease and cancer. Far too little has been spent in research into the reasons why so many children are deprived of the opportunity of enjoying even the span of years we are now trying to prolong. The Children's Medical Research Foundation will be established to meet this urgent need, and I most earnestly commend the appeal for funds for its support to every thoughtful member of the community.

This is the type of appeal in which an approach to sentiment as well as to reason may quite legitimately be made, and no doubt the general appeal to the public will be handled competently with this in mind. However, so far as the medical profession is concerned, there should be no necessity to "sell" the appeal at all. Its merits and urgency will be apparent to all thoughtful doctors, and we look confidently to them for a generous response. Donations may be sent to the Treasurer, Children's Medical Research Foundation Appeal, Royal Alexandra Hospital for Children, Camperdown, N.S.W. They will be formally acknowledged in the Press.

Current Comment.

MOUTH-TO-MOUTH RESUSCITATION (EXPIRED AIR INFLATION).

> And he went up, and lay upon the child, and put his mouth upon his mouth, and his eyes upon his eyes, and his hands upon his hands: and he stretched himself upon the child; and the flesh of the child waxed warm. (II Kings, iv : 34.)

So was described the method for resuscitation of a child used by the Hebrew prophet Elisha two thousand years ago. It is interesting to note that what is essentially the same method has again become favoured. A great many investigations have been made to determine the best method of artificial respiration for resuscitation, and various manual methods have at different times been favoured. The National Academy of Sciences—National Research Council in the United States—has held several discussions on the subject. The most recent of these, on March 8, 1957, was called to consider new evidence. The panel selected consisted of eight men from universities, research institutes, hospitals and the army, all of whom could claim to know a great deal about artificial respiration. They were assisted by a number of skilled experimentalists. The findings of the panel have been summarized by one of its members, D. B. Dill, the Deputy Director of Medical Research, U.S. Army Chemical Warfare Laboratories, and this with three papers contributed to the symposium is published in *The Journal of the American Medical Association*, May 17, 1958.

After considering all the evidence, the members of the panel were of the unanimous opinion that the mouth-to-mouth method of resuscitation for infants and small children was preferable to the rocking method, and to the back-pressure, arm-lift and other manual methods. They recommended that the American National Red Cross should, in the forthcoming revision of its first-aid manual, include a description of mouth-to-mouth resuscitation, following a preliminary manœuvre to clear the airway, for emergency resuscitation of infants and small children. They recommended also that "all possible media of public information be utilized to disseminate information concerning the use of this method". The method to be adopted, as reported by Dill, is as follows:

> Step I. Clear the mouth of any foreign matter with the middle finger of one hand. With the same finger hold the tongue forward.
>
> Step II. Now place the child in a face down, head down position and pat him firmly on the back with the free hand. This should help dislodge any foreign objects in the air passage.
>
> Step III. Place the child on his back and use the middle fingers of both hands to lift the lower jaw from beneath and behind so that it juts out.

Step IV. Hold the jaw in the position described in Step III, using one hand only.

Step V. Put your mouth over the child's mouth and nose, making a relatively leak-proof seal, and breathe into the child with a smooth steady action until you observe the chest to rise. As you start this action move your free hand to the child's abdomen, between the navel and the ribs, and apply continuous moderate pressure to prevent the stomach from becoming filled with air.

Step VI. When the lungs have been inflated, remove your lips from the child's mouth and nose and allow the lungs to empty.

Repeat this cycle, keeping one hand beneath the jaw and the other hand pressing on the stomach at all times. Continue at a rate of about 20 cycles per minute. After every 20 cycles you should rest long enough to take one deep breath. If at any time you feel resistance to your breathing into the child and the chest does not rise, repeat Step II, then quickly resume mouth to mouth breathing.

Of the individual contributors to the discussion, A. S. Gordon and four others, in a paper on "Mouth-to-Mouth versus Manual Artificial Respiration for Children and Adults", give details of their experimental observations. Studies were made on normal infants and young children. They were anæsthetized and rendered temporarily apnœic from hyperventilation and small doses of succinylcholine, and a series of methods of manual artificial respiration and mouth-to-mouth breathing were performed on them. Measurements were made in various ways to determine the tidal volumes and ventilation with and without the use of an endotracheal tube. When the endotracheal tube was used to ensure an unobstructed airway, mouth-to-mouth insufflation always provided maximal pulmonary ventilation. This was between two and three times the normal resting tidal volumes. The three manual methods yielded comparable amounts of ventilation, which was only one-half to two-thirds as much as that produced by mouth-to-mouth breathing. Manual rocking methods gave inferior results. Mouth-to-mouth resuscitation gave essentially the same ventilation with and without the endotracheal tube. Only one-half of the subjects were adequately ventilated by the manual methods when artificial means were not used to give a free airway. Gordon and his colleagues state that "their studies demonstrate the unequivocal superiority of mouth-to-mouth breathing over all manual methods of resuscitation for infants and small children". With normal respiration, expired air contains about 16% of oxygen and 4% of carbon dioxide. The first 150 cubic centimetres of the rescuer's expired air is the dead-space air, which is practically inspired air. This dead-space air is the first gas exhaled by the rescuer into the child's lungs, and in children up to two or three years of age this will constitute their entire tidal volume. Even in older children and adults the hyperventilation provides more oxygen in the lungs than normal and very little increase in carbon dioxide concentration. Experiments on adults rendered apnœic by succinylcholine while under general anæsthesia gave essentially the same results as with children. Slight variations in the method are necessary, such as pulling the jaw forward with the thumb of one hand in the mouth and clamping the nostrils with the other hand while the rescuer puts his mouth directly over the patient's mouth. As there is no hand available to keep continuous pressure over the stomach, this must be attended to periodically or undertaken by another rescuer. A mask may be placed over the patient's mouth or breathing may be carried on through an endotracheal tube.

J. O. Elam and three others, in a paper on "Oxygen and Carbon Dioxide Exchange and Energy Cost of Expired Air Resuscitation", confirm the results obtained by Gordon *et alii* and give much more detail of the gaseous exchanges. They found that by use of this method the blood was adequately oxygenated after four breaths. The data reported in their paper refute the widespread prejudice that a rescuer's exhaled air contains too little oxygen and too much carbon dioxide.

P. Safar, in a paper on "Ventilatory Efficacy of Mouth-to-Mouth Artificial Respiration", completely supports the claims and findings of the other observers. He writes: "Our data indicate clearly that the inspired air inflation methods should be taught for general use in both adults and children."

ASPIRIN AND A.P.C. IRRITATION OF THE STOMACH.

ASSESSMENT of the value of drugs and of their effects on different organs is beset with difficulties, even when we are considering such a widely used drug as acetylsalicylic acid. It has been claimed that aspirin, either alone or in A.P.C. mixtures, often causes severe irritation of the stomach, and several preparations are available in which the aspirin has been buffered. It is claimed that the buffered preparations do not cause stomach irritation. In a comment in these columns on May 3, 1958, a paper by G. A. Cronk was reviewed in which the claim was made that there was no advantage gained by buffering aspirin. The investigation appeared to have been carried out very carefully, with the precautions which are now considered necessary for such an investigation. On the other hand, at the World Congress of Gastroenterology held in Washington in May, 1958, David B. Sher presented a paper in which he claimed that aspirin buffered with aluminium glycinate and magnesium carbonate was "extremely well tolerated" by subjects who react adversely to straight aspirin or A.P.C. products. In this investigation extraordinary precautions were taken to prevent the subject from knowing what preparation was given at any time and what symptoms were looked for. A total of 236 subjects with a prior history of gastric irritation following the taking of unbuffered aspirin were investigated. Of these 94% experienced gastric reactions to the test products on one or more occasions. Gastric reactions were encountered after the taking of buffered aspirin in some cases, but the unbuffered products were responsible for 97% of gastric reactions lasting more than 25 minutes. Severe reactions were encountered more frequently with A.P.C. products (33%) and aspirin (18·3%) than with the buffered aspirin (3·9%). The presence of food in the stomach appeared to offer no protection against aspirin or A.P.C. irritation. It will be noted that the subjects of this investigation had all complained of gastric irritation following the intake of aspirin or A.P.C. preparations prior to this investigation, whereas the subjects of Cronk's investigation were unselected in this regard.

Several other papers on this subject have appeared over the past few years, but they show various deficiencies in design or execution of the investigation. Sher has put up a very strong case for the use of buffered aspirin in those patients who show gastric disturbances in association with the taking of unbuffered aspirin, but he gives no evidence that the great majority of patients would gain any advantage from the buffering.

SURVEY OF RUBELLA PREGNANCIES.

THE survey of rubella pregnancies to which we have referred previously[1] is continuing at the Department of Obstetrics and Gynæcology in the University of Melbourne. Through the cooperation of practitioners, over sixty cases of rubella in the first trimester have so far been collected. Early results so far tend to confirm figures given in a recent American paper by Greenberg, who reports a first-trimester malformation rate of 12%. However, more cases are required to arrive at a statistically significant figure for Australia, and practitioners are urged to report further cases, which are expected to occur in the coming spring epidemic. Reporting should be done on the same day as the rubella is first seen, by telephone or telegraph, to Dr. David Pitt at the above department (FJ 0484) or at his home (BL 7392).

[1] M. J. AUSTRALIA, 1957, 2: 801 (November 30).

Abstracts from Medical Literature.

HYGIENE.

Leprosy.

SIR LEONARD ROGERS (*Lancet*, July 19, 1958) surveys the progress in the control of leprosy since, in 1917, he recorded that a number of early cases of leprosy had been cleared of active symptoms by the injection of a new soluble hydnocarpate of soda. Up to that time the only plan in common use for controlling leprosy was the compulsory segregation for life of all discovered cases. The introduction of the sulphones in the 1940's was another big advance, and with the advent of effective therapy and the institution of a more realistic administrative approach great progress has been made. However, the problem is still vast. This paper deals particularly with leprosy in British-controlled tropical African territories, where it is estimated that there are still over 700,000 people suffering from leprosy, of which about 130,000 are receiving treatment. The author concludes by stating that the way has been prepared for the steady reduction in the incidence of leprosy, probably nearly to the point of eradication, if only the necessary staff and funds are forthcoming.

Nursery Staphylococcal Infections.

W. MURRAY, G. MCDANIEL AND M. REED (*Am. J. Pub. Health*, March, 1958) confirm the value of the telephone survey in supplementing information obtained from interviews of physicians in an epidemiological survey of an outbreak of staphylococcal infection in an American city. After the deaths of two infants from staphylococcal infections, all hospital records were investigated. These revealed a number of staphylococcal infections in infants and mothers while in hospital. Physicians were then phoned and it was found that further infections had occurred in mothers and infants after leaving hospital. The incidence indicated by these two investigations was an attack rate of 3·9% of 277 mothers delivered during a three-month period, and of 9·3% of their 279 infants. A telephone survey of one-third of the mothers delivered during this period revealed an attack rate twice that indicated by the first two investigations. Only four of the 51 infections were detected in the hospital nursery. Mothers and infants who subsequently became infected had remained in hospital longer than those who escaped infection. The telephone survey proved to be an accurate, non-provocative epidemiological tool for retrospective case finding, requiring less time and providing easier cross-checking than interrogation of physicians.

R. FEKETY *et alii* (*Am. J. Pub. Health*, March, 1958) describe an epidemic of nursery-derived suppurative illnesses of infants and mothers due to *Staphylococcus aureus* phage type 52/42B/80/81. Of all deliveries during the epidemic, 10% were complicated by disease due to the organism; one-half of the infections began after the infant was discharged from the hospital. Infant pyoderma was the most common disease noted; breast abscesses were the most common maternal infection. Most of the infections were mild, but several infant fatalities occurred. The strain was recovered from infants, nurses, mothers, and nursery dust. The epidemic was apparently controlled by the removal of carriers from the nursery, by the application of more rigid aseptic techniques, and by measures designed to prevent the spread of the organism from infant to infant. Evidence is presented suggesting that this strain, hitherto considered a "hospital staphylococcus", is now commonly found in the general community. The epidemiology and control of nursery-derived staphylococcal infections are discussed.

Public Health Educators.

R. A. BOWMAN (*Am. J. Pub. Health*, February, 1958) investigated the relationships between the actual and stated duties and responsibilities of public health educators and their recommended qualifications and the preparation courses offered by selected schools of public health. The aim of the investigation was to clarify the role of public health educators in health agencies. Details of the investigation are given and the author concludes that the findings of the investigation indicate that no definite pattern of consistent relationships exists between the work done by the selected public health educators, the requirements for positions in health education, the educational preparation offered in the schools of public health and the American Public Health Association's statement of educational qualifications.

Dermatitis in Industry.

D. C. BRAUN AND R. SITGREAVES (*Arch. Indust. Hyg.*, April, 1958) report on an investigation of dermatitis in a cross-section of American industrial organizations, consisting of 36 companies with 103,586 employees in five different regions and representing eight different manufacturing categories, to determine the incidence of occupational dermatitis, and in particular dermatitis due to oil. The over-all rate of occupational dermatitis for all workers was 0·8% per annum. That due to oil was 0·3% per annum. The over-all rate for production workers was 1·2%. More dermatitis was reported from large factories than small. This may be due to better medical facilities for diagnosis in large factories. Women had a higher incidence rate than men. The results indicated that dermatitis rates were not higher in warm and sunny climates. No significant seasonal variation was apparent. Cutting oil accounted for 69% of cases of dermatitis due to contact with oil.

Weight Control.

H. J. KENNEALLY (*Am. J. Pub. Health*, February, 1958) gives details of a pilot study in weight control by a group of individuals rather than by each individual on his own. The Oregon Heart Association had decided that a programme aimed at the prevention of overweight could have an important part in the reduction of mortality and morbidity from cardiovascular disease. A programme of weight control by group methods was planned. Details of methods used in the pilot study are given, and at the end of the study the following conclusions were made. The average participant can lose weight successfully in a programme that applies group methods. The amount of weight lost by the average participant was 10 pounds. The amount of weight lost by the average participant was proportionate to the number of meetings attended. The age of the individual participant was a factor in both the amount of weight lost and the number of meetings attended. The older participants lost less weight and attended less meetings than the younger ones. Plans should be formulated for continuation of the groups, under their own leadership, after completion of the formal course. The more homogeneous the group, the greater the weight loss per participant, the better the average attendance, and the lewer failures to complete the course. It was concluded that the programme had merit, and the author was instructed to proceed with plans for a state-wide programme of weight control that would utilize the procedures and findings of the Portland pilot study.

Some Inquiries into the Toxicology of Zinc Stearate.

H. E. HARDING (*Brit. J. Indust. Med.*, April, 1958) reviews a number of reports on the effect of inhalation and ingestion by human beings of zinc stearate. He also gives the results of experiments involving the injection of a suspension of zinc stearate into the lungs and peritoneal cavities of rats and cavies, respectively. Zinc stearate is used commercially as a substitute for talc in the manufacture of rubber articles to prevent them sticking together. From the experiments it appears that zinc stearate in suspension in tap water or in a mixture of skimmed milk and physiological saline is acutely irritating, and if injected through the larynx into the lungs of rats in doses of 950 milligrammes kills nearly half of them. Death results from acute œdema of the lungs. Given intraperitoneally to cavies, 100 milligrammes killed two out of six animals within a short time, but 50 milligrammes were tolerated well. Zinc stearate disappears within a period of about a fortnight from the lungs of rats, presumably by processes of phagocytosis and solution. No fibrosis results in the lungs of these animals after a single injection through the larynx. In the peritoneum of cavies the powder provokes a granulomatous reaction, at first with a polymorphonuclear leucocytic response. These cells die and are surrounded, and eventually digested, by histiocytes. The stearate remains visible in the peritoneum of cavies much longer than in the lungs of rats, possibly because it is rapidly aggregated into masses, and it can still be found after 42 days enclosed in an envelope of histiocytes, outside which is a narrow zone of fibroblastic activity. However, somewhere between the forty-second and the one hundred and tenth days the stearate disappears and the granulomatous process regresses until no sign of it can be found. These experiments, while suggesting that inhalation of zinc stearate might produce acute

inflammation of the upper respiratory tract of the lungs, provide no evidence of any permanent fibrogenic effect.

PATHOLOGY.

Cytological Examination of Urinary Sediment.

N. C. FOOT, G. N. PAPANICOLAOU, *et alii* (*Cancer*, January-February, 1958) have reviewed 2829 cases in which urinary sediment was examined for cancer cells between the years 1945 and 1954. The highest degree of accuracy was obtained for tumours of the bladder, ureter or renal pelvis. Of 212 patients with these tumours, a correct diagnosis was made from examination of the urinary sediment in 62%; in six other cases an incorrect "positive" diagnosis was made. Of patients with tumours of the renal parenchyma, in only 8% was a positive report returned, and there were five "false positives". Of those with carcinoma of the prostate, 15% were correctly diagnosed, and the over-all false positive rate was 1% (six cases). The authors consider that prostatic smears may give a higher rate of success in diagnosing carcinoma of the prostate than urinary sediment. Confusion in cytological interpretations was caused in a few instances by renal calculus and chronic cystitis.

Carcinoma of Liver and "Thorotrast".

A. D. MORGAN, W. H. W. JAYNE AND D. MARRACK (*J. Clin. Path.*, January, 1958) describe a case of primary liver carcinoma occurring 24 years after the intravenous injection of "Thorotrast". The methods of assessing the residual radioactivity in the organs obtained at necropsy are described. The liver was not cirrhotic and the mechanism of carcinogenesis is thought to have been a direct action on the liver cells. The authors have reviewed the literature and find that malignant growths of the liver after "Thorotrast" administration fall into two broad groups: malignant hæmangioendotheliomata on the one hand and primary liver-cell or bile-duct carcinoma on the other.

Transplacental Bleeding from the Fœtus.

H. B. GOODALL *et alii* (*J. Clin. Path.*, May, 1958) describe two examples of bleeding of the fœtus into the maternal circulation and the methods used to establish the diagnosis. They point out that the anæmic infant may be misdiagnosed as suffering from asphyxia pallida or intracranial injury when it is actually in post-hæmorrhagic shock. Rapid diagnosis and prompt blood transfusion may save lives in these cases.

Cancer of the Stomach in Hiroshima.

E. S. MURPHY AND A. YASUDA (*Am. J. Path.*, May-June, 1958) have compared the occurrence in Hiroshima of carcinoma of the stomach among individuals who had been exposed to the atom bomb with that among those who had not been exposed. Carcinoma of the stomach has a high incidence in Japan, and it was selected for this reason. There were 535 cases of gastric carcinoma recorded in the pathology records of the Hiroshima Atomic Bomb Casualty Commission between December, 1948, and June, 1957. The incidence and behaviour of gastric carcinoma among those exposed to the atom bomb explosion did not differ significantly from its incidence and behaviour among those not exposed.

Congenital Lupus Erythematosus.

G. R. HOGG (*Am. J. Clin. Path.*, December, 1957) reports a case of acute disseminated lupus erythematosus associated with subendocardial fibroelastosis in a newborn infant whose mother was suffering from subacute lupus erythematosus. The author suggests that subendocardial fibroelastosis may be a collagen disease and that in some previous studies hæmatoxylin bodies have been incorrectly interpreted as calcium because of the similarity of their staining with hæmatoxylin.

Kerato-Acanthoma.

T. N. GLADIALLY (*J. Path. Bact.*, April, 1958) has found that there is a striking morphological similarity between kerato-acanthoma in man and the self-healing lesions which occur in the skin of rabbits treated with chemical carcinogens. The lesions seem to arise in pilo-sebaceous follicles, and in both man and the rabbit the lesions have a brief period of rapid growth followed by slow involution. The possibility of carcinogen induction of the human growth is discussed and there is an extensive histological description of the lesions.

Fat Embolism.

H. E. EMSON (*J. Clin. Path.*, January, 1958) reports that he has demonstrated fat embolism in 89% of patients dying after injury. Pulmonary fat embolism always accompanies systemic fat embolism, but even when severe it is not of importance as a cause of death. Although only in 3% of cases was fat embolism the chief cause of death and in another 4% was it a contributory cause, cerebral fat embolism is thought by the author to be more important as a cause of symptoms and death than is commonly realized. Cerebral fat embolism does not always give rise to the syndrome usually described as characteristic of this condition.

Experimental Fibrinoid Lesions.

P. E. FEHR AND J. G. BRUNSON (*Am. J. Path.*, November-December, 1957) state that the common characteristic alteration in the group of diseases which are known as collagen diseases, is the presence of diffuse vascular fibrinoid lesions, associated with areas of hæmorrhage and necrosis in many organs. One central problem in the pathogenesis of these diseases is the origin and nature of this fibrinoid material. The authors state that they have produced fibrinoid lesions in the heart, lungs, spleen, liver and kidneys of rabbits which were given Gram-negative endotoxin or sodium polyanethol sulphonate ("Liquoid"), 72 hours after a six-day course of bovine gamma globulin injections. The coronary arterial lesions resembled those of polyarteritis and in the kidney fibrinoid was deposited focally in glomeruli and in the media of the large renal arteries. Proliferative glomerulitis was also present. Electrophoretic studies of serum proteins were also carried out. There was, however, no correlation between the changes found and the incidence of fibrinoid lesions.

Cerebral Thromboangiitis Obliterans.

L. WOLMAN (*J. Clin. Path.*, March, 1958) reports the case of a young man with cerebral manifestations of thromboangiitis obliterans with no discoverable peripheral vascular involvement. The clinical features are correlated with the pathological findings. There is a review of the literature of cerebral Buerger's disease, and the author stresses the frequency with which this may precede limb involvement by varying periods up to 20 years. A diagnosis of the condition should always be suspected when there is cerebral thrombosis in a young adult in whom heart disease, arterial degeneration, generalized systemic disease and demyelinating diseases can be excluded.

Sarcoid Reaction Associated with Carcinoma.

G. A. GRESHAM AND A. G. ACKERLEY (*J. Clin. Path.*, May, 1958) describe a variety of granulomatous reactions in lymph nodes related to, but distinct from malignant neoplasms. This picture was found in 10 cases of carcinoma, of which four were in the stomach, and in one case in which the lesions were an incidental finding at necropsy in a case of myocardial infarction. They classify the lesions into those resembling sarcoidosis and those resembling Stengel-Wolbach's sclerosis. The pathogenesis of the lesions remains unknown.

Pulmonary Thrombosis in Cyanotic Heart Disease.

P. V. BEST AND D. HEATH (*J. Path. & Bact.*, April, 1958) confirm the conclusion of Rich that a reduced rate of pulmonary blood flow and increased blood viscosity are the important ætiological factors in pulmonary thrombosis affecting patients with cyanotic heart disease. They find a very high incidence of thrombosis when there had been a low pulmonary blood-flow and severe polycythæmia, but in patients with a high flow and slight polycythæmia thromboses were much fewer. The characteristic histological picture of the vessels in the lungs in cases of cyanotic heart disease without hypertension are, in addition to thromboses, pulmonary arterial hypoplasia and the development of collateral circulation.

Renal Lesions Produced by Corticoids.

S. A. BENCOSME, D. L. WILSON AND D. A. ROSEN (*Arch. Path.*, March, 1958) have found that prednisone and prednisolone each produce lesions in the rabbit's kidney which are indistinguishable from those produced by cortisone. These lesions resemble the lesions of diabetic glomerulosclerosis in the human, but in the rabbit the lesions are the same whether the animal is rendered diabetic or not and are uninfluenced by hypertension.

Congresses.

AUSTRALIAN PHYSIOTHERAPY ASSOCIATION.

THE seventh biennial congress of the Australian Physiotherapy Association was held at Perth, Western Australia, from May 25 to 31, 1958.

Official Opening.

The congress was opened by His Excellency the Governor, Sir Charles Gairdner, the Patron of the Western Australian Branch. The Federal President of the Association, Dr. W. N. Gilmour, welcomed the visitors to the congress and asked them to criticize freely, as criticism led to improvement. He stressed the importance of orthodoxy with its use of proved methods and constant effort to improve those methods. Heterodoxy he defined as the using of new, untried methods without due care and consideration. He emphasized the importance of a high ethical standard, and pointed out that the community enjoyed security through the knowledge that physiotherapists had a safe standard of treatment and conformity to ethics.

Inaugural Address.

The inaugural address was given by Professor E. G. Saint on the subject "Education for the Future". He reviewed the present methods of education for medical and medical ancillary students, suggesting that there was too much emphasis on special subjects appertaining to medical courses at too early an age; as a result the future student was channelled into a narrow canal instead of having a wider general training before specializing. He also thought that it would be of advantage if all members of ancillary services, such as physiotherapists, occupational therapists and dietitians, had basic training in the same centre, specializing towards the end of their course just as medical students all took university degrees of bachelor of medicine and surgery before going on to specialize. He considered that a medical ancillary training centre would have the same effect as a residential college in a university, mixing all types of student, giving them insight into other types of training and causing a sympathetic understanding of problems.

Treatment of Pain in the Upper Half of the Body.

Miss R. Humphery (New South Wales), in a paper entitled "A New Principle for the Treatment of Pain in the Upper Half of the Body", explained that she classified the muscles which moved the shoulders, neck, arms and hands as horizontal and those of the legs, hips and trunk as longitudinal. She was convinced that pain in horizontal muscles, which was referred to in different areas as fibrositis, arthritis, neuritis, tennis elbow and migraine, was caused by some imbalance between the flexors and extensors of the hand and forearm and was referred from the periphery inwards, not, as was usually stated, from the centre outwards. She advocated the use of deep massage, mainly the Cyriax type of friction, to certain spots, followed by extension exercises to the fingers, arms and shoulders. She then demonstrated her technique on a model.

Paraplegia.

A discussion and demonstration relating to the management of paraplegia were presented by Dr. G. M. Bedbrook, Director of the Paraplegic Unit, Royal Perth Hospital, with Mr. Allan Panton, Miss W. MacClure, Miss Fay Sharp, Miss Junette Shepherd, Mr. L. J. Johnson and Mr. W. E. Fletcher.

Dr. Bedbrook briefly described the causes of paraplegia, blaming accidents for a large proportion of cases. He discussed the patients' disabilities and their management, emphasizing the need for accurately assessing disability, keeping a careful watch for recovery if and when it occurred, and maintaining mobility of joints. Deep breathing, voluntary or assisted, must be kept up and the optimum posture assured.

A demonstration followed of the assessment and initial treatment of a patient with an acute spinal cord lesion. Breathing voluntarily and with a cough machine formed the start of treatment. The patient was then postured in the hyperextension position. A full range of passive movements was given to discover the extent of spasm and its severity. When active movements were manifest, a comprehensive muscle chart was completed; signs of recovery would be charted as they showed. Free exercises with slings and springs and resisted movements with weights were then carried out. As soon as possible weights and pulley circuits were introduced and the weights consistently increased. Finally, electrical stimulation was given for the preservation of muscle power.

Miss Sharp and Miss Shepherd demonstrated the class system of bed exercises. It was found that the patients benefited from the competition, and as they were encouraged to do everything possible for themselves during the class without assistance, they were able to carry on their exercises by themselves when they left hospital. All beds were fitted with overhead apparatus of bars with handles and pulleys, so that the patient could assist himself in moving about the bed. The class began with the patients lying on their backs pulling weights attached to ropes hung over pulleys followed by pull-ups on the overhead bars. Designed to strengthen the muscles of the trunk and upper limbs, the exercises progressed gradually. Mobility of lower limbs and prevention of contractures were achieved by the patient's use of pulleys for passive movements of the legs. Slings and springs were employed to support limbs and to prevent friction. Active loosening and relaxation exercises for arms and shoulders were included, quadriplegics joining in where they could. Localized breathing and breathing exercises were given for general health and to assist in prevention of respiratory disorders. The paraplegics then turned themselves over into the prone position and did hyperextension exercises to strengthen the muscles of the upper part of the trunk. Throughout the class a careful check was kept on posture, whether sitting or lying.

Patients then demonstrated standing, walking and mat-mobility, which formed the next stages of treatment and were considered of great importance to the patient for overcoming vasomotor disturbances and obtaining postural sensibility and improvement of bladder and bowel function.

Finally, demonstrations were given of basketball, softball, dry-fly casting, fencing, swimming, table tennis, putting the shot, javelin throwing and weight lifting by the patients. Mr. Fletcher, who was in charge of this section, emphasized the importance of teamwork with staff and patients.

The Anatomy of Pain.

In a discussion on the anatomy of pain, Professor David Sinclair explained that the original description of pain was "the opposite of pleasure"; there was no idea of other modalities. When the innervation of the skin was realized, it led to over-enthusiasm, and innumerable small nodes or ganglia were named after their discoverers. Professor Sinclair referred to the theory evolved by Max von Frey that nerve endings had separate functions from nerve fibres, and to the idea that the bead-like nerve endings in the skin were concerned with pain, as areas insensitive to warmth or touch had only the bead-ended plexus. He pointed out two facts that were against the theory; hairy skin was sensitive to other things than pain, and the cornea of the eye was sensitive to temperature and pain, yet both areas had only the bead nerve endings. Current thought seemed to incline to the theory that less sensitive areas had a wider mesh of nerve fibres; but where a lily-pad design of overlapping fibres occurred there was acute sensitivity. He went on to explain that there were no different types of nerve fibres; they were only large or small. The large were probably the fibres of touch and the small those of pain. Temperature fibres were unknown as yet. It had been supposed that different impulses travelled by separate pathways on reaching the spinal cord, but Spiller's operations of tractotomy had made it clear that that was not so. The most recent discovery had been the difference between perception and appreciation of pain, which had not been realized until it was found that removal of the cortex would remove the appreciation of pain; the patient would continue to suffer pain, but would be quite unconcerned about it.

Manipulation in the Treatment of Joint Injuries.

A debate was held on the proposition: "That manipulation has no place in the treatment of injuries to the knee and shoulder joints." Speaking for the affirmative were Dr. F. G. Bell and Miss S. M. Seward. Speaking for the negative were Dr. P. I. Cromack and Miss M. Cummings.

Dr. Bell defined manipulation as a forced movement, with or without anæsthesia, beyond the patient's control. He said that, except for the reduction of a dislocation, complete joint rest was essential to allow healing of the soft structures. In knee injuries, frequent manipulations tended to rupture those structures and, in extreme cases, even the quadriceps tendon. In shoulder injuries, such as a frozen shoulder, the rotator cuff and biceps tendon, which were avascular, tended to degenerate easily; further damage was

done to those by manipulation. Healing was slow, and the best treatment was by means of injections of hydrocortisone to ease the pain, complete rest until the soft structures were healed, and then static contractions and faradism.

Miss Cummings defined manipulation as the application of controlled force. She gave a short history of manipulation from its origin in 1745, when John Hunter at the College of Surgeons discovered that the circulation in the tissues around joints benefited by movement. Joints were rested until the pain and swelling had subsided. Manipulations were then attempted, which resulted in the breakdown of adhesions with consequent renewal of pain and swelling. Through lack of pathological knowledge that was not understood, and the patient was rested again. The system lasted for a century and a half, notably because surgeons feared the risk of injury to tuberculous joints. Miss Cummings listed the objects of manipulations as: (i) loosening of periarticular adhesions, (ii) improvement of functional or hysterical contractures of joints, (iii) reduction of dislocations and subluxations, (iv) breakdown of adhesions in muscles and tendons.

Miss Seward stressed the importance of accurate diagnosis of all injuries and emphasized the adverse psychological effect of manipulation with or without anæsthesia. She summarized the argument thus: (i) In acute injuries manipulation had no place except where bony structures had been displaced; strain of ligaments and tearing of cartilage delayed healing of soft tissues. (ii) Manipulation of locked joints caused further damage of the articular cartilage. (iii) Manipulation forced mobility of joints beyond the limit of pain, resulting in hæmorrhagic effusion, which could further limit subsequent movement; that position might be altered by the use of hydrocortisone, but as yet it was too early to make an accurate assessment.

Mr. Cromack maintained that manipulation formed an integral part of orthopædics, but had been neglected by the medical profession and had fallen into the hands of unqualified practitioners. Accurate diagnosis was possible, and there were five causes of limitation of movement: muscular spasm; adhesions, extraarticular and intraarticular; bony blocks; a combination of those already mentioned; ankylosis. All those conditions benefited from manipulations, whether under anæsthesia or not, although the latter was essential in the case of shoulder dislocation or trauma.

After the discussion, the chairman suggested that the subject would never be settled and quoted several English authorities for both sides. He personally considered the timing of manipulation of paramount importance.

Rheumatoid Arthritis.

Dr. Phyllis Goatcher read a paper on the modern aspects of rheumatoid arthritis. She said that rheumatoid arthritis was the Cinderella of diseases; its cause was unknown, its treatment empirical. The old theory of causation was that there was a local centre of infection such as teeth or tonsils, but it had been found that the removal of the infected focus was of no benefit and the removal of teeth was more likely to be bad for the patient from the nutritional aspect. Dr. Goatcher outlined the current approaches to the disease, stressing the importance of precise early diagnosis.

Dr. Goatcher then went on to discuss the management of patients with rheumatoid arthritis. She said that physiotherapy was expensive, as were many drugs, but amongst the latter "Butazolidin" and cortisone were very helpful. Rest was essential. For the relief of pain two to four tablets of aspirin were given four-hourly; "Butazolidin" was an analgesic, but it had dangerous side effects. Cortisone used as a local injection assisted 50% of patients. Gold had predictable complications, but must be used in the first five years and in conjunction with general treatment. Physiotherapy should be directed to the general care of the patient and not to isolated spots. In Western Australia rehabilitation was poor; there was little hospital-home liaison, and no sheltered workshops were available, so that the patient often had no occupation to keep his mind off pain and disability.

Polythene and Polyeurothene Splints.

Mr. T. J. Lyall demonstrated the use of polythene and polyeurothene in the making of splints. He said that in a department there should be suitable heating apparatus of the oven type, but went on to show that an electric frypan could be used to heat the two materials and fuse them for splints. Using a model, he showed how to cut a paper pattern of the required size and shape, cut the polythene and polyeurothene splints from this, heat them, roll them into complete fusion, mould them to the patient's limb, correcting deformities where necessary, and then, after the splint was set, fix straps to fasten it and smooth rough edges. The splints were light and rigid, giving firm support but not impeding movement of adjacent joints.

Manipulation for Cervical Lesions.

A demonstration of treatment of lesions of the cervical region by manipulation was given by Mr. G. D. Maitland (South Australia). Using a model, he demonstrated types of manipulations and the best grip with which to carry them out, stressing that little movements performed frequently were more effective than violent ones performed only once or twice. He followed this with a film of some of his treatments, which showed even more clearly the points he wished to emphasize.

The Development of Thoracic and Cardiac Surgery.

Dr. J. A. Simpson discussed the development of thoracic and cardiac surgery. He described the last twenty or thirty years as the most exciting in thoracic surgery; most of its pioneers were still living. Tracing the development of surgery in general, he explained that in ancient Greece medical men were general practitioners, but by the Middle Ages medicine had been divided into two groups by a horizontal line. Above the line were the physicians, who tended to become more and more theoretical and hired a lower order of men, who became known as barber-surgeons and were of a doubtful standard. Progress became impossible until the fifteenth century, when licences were granted to the less clever medical students to practise surgery but not medicine. Operations were mostly amputations and opening of abscesses, with alcohol as the only anæsthetic. In the second half of the nineteenth century, Lister introduced antiseptic surgery, and aseptic methods followed. General anæsthesia appeared about the same time and opened a wide field of surgery. Thoracoplasty was performed simultaneously in Germany and America by Sauerbruch and Alexander. They were followed by Holst and Semb, of Oslo.

Dr. Simpson said that cardiac wound suture, attempted in 1895, remained the only form of cardiac surgery until the 1930s. The delay had three main causes: (i) lack of knowledge of physiology, (ii) lack of knowledge of anæsthesia, (iii) the attitude of physicians to surgery. However, the horizontal division was disappearing and a vertical one appearing in which certain branches of surgery were handled by specialists. In 1939 Gross ligated the ductus arteriosus. Between 1939 and 1948 external cardiac surgery was performed. Between 1948 and 1953 blind intracardiac surgery came into use. In 1953 operations on the open heart became possible through the discovery of hibernation anæsthesia (hypothermia); but as that could be of ten minutes' duration only, the field was limited. Next came the development of the mechanical heart. Summing up, Dr. Simpson said that thoracic surgery was well established. Surgery of the open heart was not yet complete, but was well on the way. The interest of the future lay in degenerative diseases. Present methods would probably be extended and heart and lung banks established in the not-too-distant future.

Treatment of Patients After Thoracic Surgery.

Miss A. Simpson (South Australia) demonstrated the treatment in bed of different types of thoracic surgical patients, the arrangement of pillows and postural drainage positions, including the most effective and comfortable position for coughing. She emphasized the importance of cooperation.

Pain in the Upper Limb.

Professor C. W. D. Lewis discussed pain in the upper limb. He began by saying that an efficient surgical team should contain at least one physiotherapist; a staff of 13 physiotherapists was not sufficient for a hospital of 570 beds. He went on to say that there were too many causes of pain in the upper limb to detail, but the main ones were pain from a local lesion, a local manifestation of a general distribution, referred pain from an intrathoracic lesion, psychogenic pain, pain originating in the region of the spinal cord, pain originating in the brachial plexus, and pain from peripheral vascular insufficiency. He then discussed the more interesting of these, in particular the scalenus syndrome and the sites of irritation in the arm, such as the elbow (medial condylar syndrome) and wrist (carpal tunnel syndrome). He said that a myelogram was useful in deciding the causes of spinal obstruction, but should not be used as a routine.

In reply to a question, Professor Lewis said that he did not consider that acroparæsthesia in pregnancy was due to faulty posture; it was a hormonal disturbance also found in menstruation.

Neurosurgery and Physiotherapy.

Dr. J. S. Lekias, in a paper entitled "Aspects of Neurosurgery and Physiotherapy", said that a neurosurgeon was no different from a general surgeon except that he dealt only with nerve tissue. Damage to those tissues was in three categories: (i) congenital and therefore related to the nerve skeleton, (ii) acquired, (iii) traumatic. In the last two, peripheral nerves were most likely to be affected. Under the second heading there were three subdivisions, neuropraxia, axonotmesis and neurotmesis. In treatment of the first two, physiotherapy played a large and important part. In the third, a deep knowledge of anatomy was required and electrical muscle tests were extremely helpful in diagnosis. Trauma to any part of the spinal cord through injury to the vertebræ usually resulted in paraplegia or quadriplegia. In such cases surgery was seldom indicated, but physiotherapy and nursing were most important.

Dr. Lekias went on to say that disk lesions were best treated by physiotherapy combined with suitable support. The brain, owing to its position and blood supply, was peculiarly affected by diseases of the blood vessels and peculiarly susceptible to trauma. Neurosurgeons owed much to anæsthetists, especially in the management of vascular disturbances inside the skull. Head injuries caused by road accidents formed the largest and hardest part of neurosurgeons' work.

Facial Palsy.

Dr. A. G. Fisher read a paper on the subject of facial palsy. He said that there were three types caused respectively by damage in the pathway above the nucleus, by damage to the nucleus and by infra-nuclear damage. In the first type electrical reactions were normal and emotional movements less affected than voluntary ones. In the second and third types all movements were equally affected and the electrical reactions were changed. The cause of Bell's palsy was unknown, but it was thought to be due to neuritis within the facial canal. There was a 94% expectation of recovery with normal faradic response and a 7% expectation of recovery with absence of this. The most successful treatment was correct splintage, interrupted galvanism and exercises.

Chronic Nervous Disease.

Miss Aura Forster discussed the planning of a programme for a person with a chronic nervous disease. She said that a very full examination was necessary, extending into a double treatment period if necessary. It should be recorded carefully under headings such as: joint range—degree and what part of range; functional activities—bed, toilet, general, eating. The latter should be classified as, for example, "adequate and independent", "needs help" (one or two persons), or "incapable". The chest movement should be assessed; it was poor in disseminated sclerosis and cerebral palsy.

Treatment must cover the prevention of deformities and provide for the interests and needs of the independent. Duration of treatment required consideration and should be divided into individual treatment of one patient by a physiotherapist, independent activity and group activity. All those must be based upon the functional activity necessary for the independence of a patient and the programme worked out accordingly; the patient should be given clear, simple, verbal explanations of how movements could be performed and where they would help. Families should be encouraged to help and should be shown how such assistance was required, with a tactful explanation that too much assistance could be harmful; the principal aim should be to make the patient sufficiently independent to be able to live at home.

Exercises for Vertigo.

Mr. J. H. Cook, assisted by final year physiotherapy students, demonstrated exercises for vertigo following such operations as fenestration. After giving a brief description of the difficulties of balance for such patients, Mr. Cook showed a variety of exercises starting with the patient in bed, moving only the eyes and head; then followed exercises in sitting, standing, walking and running, some done with the eyes open, some with them closed, some combining eye movements with those of other parts of the body, gradually increasing in activity and coordination.

Care of the Injured Hand.

Dr. H. K. McComb discussed the care of the injured hand. As a plastic surgeon, his interest lay in the repair of hand injuries, and he used slides in colour and in black and white to illustrate the subject. He described the various types of injury caused by road or industrial accidents or occupational risks and how they were best repaired. He said that function was the main aim, and that it was better to amputate a finger than leave a flabby stump which got in the way. He showed different methods of achieving that so as to give the patient a useful pincer-grip. He also demonstrated diagrams of tendon transplants and photographs of injured hands before and after repair, together with those in various stages of operations. Finally, he showed a colour film of different types of skin and the uses of each. The film included pictures of patients undergoing treatment and showed how their disability was overcome. In some cases they were able to return to their previous occupation unhandicapped.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE NEW CENTRAL BOARD OF HEALTH OF VICTORIA.[1]

[From the *Australasian Medical Gazette*, February, 1890.]

THE new Central Board of Health as elected under the recently passed Health Act of the Colony is for the purpose for which it is called into existence a remarkable body. Of its nine members but one is a medical man, and he is new to Australia, in fact he has not arrived here yet. The other members are for the most part shire councillors or aldermen, the chairman being a barrister. In commenting on his appointment The Argus says that as he has been in the habit of controlling "a somewhat demonstrative Assembly—the University Senate—he should be able to keep the Board of Health under due control". It thus seems to be thought probable that there are likely to be lively times. The success or failure of a board of health is a matter of infinitely greater personal interest to the lay people than to the medical profession except in so far as its failure will mean more disease, increased professional work, and consequent profit to its members. Such a body, composed with one exception entirely of laymen, is of so exceptional a character that we shall wait in amused expectation for the probable exhibition which it will make of its incapacity.

Correspondence.

THE INCIDENCE OF LEUKÆMIA IN AUSTRALIA.

SIR: I am indebted to Dr. E. V. Keogh for drawing my attention to an error in my interpretation of Lancaster's data on the incidence of leukæmia in my paper in the August 2, 1958, issue of your Journal. The correct form of my table is as follows:

TABLE II.

The Incidence of Leukæmia in Australia.

Period.	Deaths per Million *per Annum*.
1908 to 1910	15
1911 to 1920	18
1921 to 1930	17
1931 to 1940	25
1941 to 1945	31
1946 to 1950	42
1951 to 1955	47

The incidence of leukæmia attributable to radiation now becomes some 10% instead of 5% of the total incidence, and the main deduction from my paper that radiation plays a small part in causing leukæmia is unaltered.

[1] From the original in the Mitchell Library, Sydney.

In writing, may I add that since my paper was completed in December, evidence has been produced (Keogh, McCall and Rankin, 1958;[1] Clemmensen and Sorensen, 1958[2]) which indicates that no significant increase in mortality from leukæmia is apparent in periods of the order of one or two decades.

Yours, etc.,

Physics Department, Peter MacCallum Clinic, Melbourne.
J. H. MARTIN.

August 11, 1958.

CHLOROQUINE.

SIR: I wonder if I can be informed as to why chloroquine has been removed from the list of free drugs and placed on the specified disease list. Many of us have for some time been using chloroquine with some success in rheumatoid arthritis and cervical spondylitis. While it is agreed that there is no logical basis for its use, I think it will also be allowed that in many cases it does provide considerable symptomatic relief. As far as I am aware, the drug is not toxic in the small doses required (520 milligrammes per day) and is not expensive (22s. per 100 dispensed). It would be interesting to know why the Commonwealth Health Department was advised to take this action. Perhaps representation could be made for its restoration.

Yours, etc.,

58 Ballarat Road, Maidstone, Victoria.
JOHN POOLMAN.

July 25, 1958.

CONCURRENT VARICELLA AND HERPES ZOSTER.

SIR: In reference to recent correspondence about concurrent varicella and herpes zoster. In June of this year a woman, aged seventy-five, was admitted to the Royal Newcastle Hospital. The illness commenced six days before admission with a painful eye, and the vesicles on her body appeared five to seven days later. She was in poor general health, having had several cerebral thromboses in the past five years, with a residual left-sided paresis. She had a fairly generalized rash of the varicella type and an acute herpes zoster ophthalmicus in the distribution of the first division of the fifth cranial nerve. The skin of this area was reddened, swollen and vesiculated, and the eyelids grossly œdematous. There was an associated iritis. She developed congestive signs in the chest with infection and loose motions, and died five days after admission.

Yours, etc.,

The Royal Newcastle Hospital, Newcastle, New South Wales.
J. ROBERTS.

August 1, 1958.

"INNOCENT" INVERSION OF THE T WAVE.

SIR: I am in entire agreement with Dr. Seldon's article on the above subject and pleased that he has brought this aspect to light. After observing that "physiological changes regarded as abnormal can lead to rejection for life assurance", he very rightly states that "this type of T wave is evidence neither for nor against the presence of ischæmic heart disease".

This waxing and waning of T waves opens up serious ground for reflection in those of us more especially engaged in the assessment of life insurance risks. Recently a proposal with a large amount at risk was presented for consideration. The applicant, aged 32, though not clinically suspect, was of mesomorphic build and of a family background with a proneness to early cardio-vascular disease. The T4 and T5 waves of his electrocardiogram alternated, during the course of a week, between normality and inversion, with or without exercise. After strenuous exertion there was no precordial distress, exercise tolerance was very good, and there was no S-T deviation in the electrocardiogram. One of the hierarchy in cardiology was co-opted and, after going to much trouble and study with further tracings, considered that the case should be heavily loaded, a view in which the writer, with minor reservations, concurred. An overseas reassurance company was approached, its reaction being towards still heavier extra premiums. Subsequently, I understand, the proposal was accepted by another company, on what terms I do not know.

After a chance discussion with a bright young colleague who had also been perplexed with these seemingly innocent T wave changes, a model of Gardberg's[1] idealized spatial QRS loop was improvised. When its shadow was projected in the frontal plane with the centre of the Einthoven triangle and zero point of the loop in one line, it was abundantly illustrative how the T vector might alter with minor changes in the heart's anatomic axes, such as might be expected after a heavy meal, exercise, emotion, smoking, deep breathing, change of posture, etc.

Thus it would seem reasonable to assume that, in some instances, T wave inversion in the outer precordial leads may be of innocent origin; that the question is not one for untutored ears nor to be resolved in life assurance practice on the principle of "All Lombard Street to a China orange".

Yours, etc.,

Sydney,
W. J. MCCRISTAL.

August 2, 1958.

[1] Keogh, E. V., McCall, C., and Rankin, D. W. (1958), "Mortality from Leukæmia in Victoria, 1946-1955", A Report from the Central Cancer Registry, Melbourne.

[2] Clemmensen, Johannes, and Sorensen, Jens (1958), "Malignant Neoplasias of Hæmopoietic and Connective Tissues in Various Countries", Danish Medical Bulletin, 5: 73.

[1] Gardberg, M. (1957), "Clinical Electrocardiography: Interpretation on a Physiological Basis".

Obituary.

ALAN VICTOR SMITH.

WE are indebted to Dr. Frank Hansman for the following appreciation of the late Dr. Alan Victor Smith.

> And fear not lest Existence closing your
> Account, and mine, should know the like no more;
> The Eternal Sāki from that Bowl has pour'd
> Millions of Bubbles like us, and will pour.
>
> —OMAR.

These are not sad but hopeful lines; they tell us that future generations will also have in the medical profession men of the calibre of Alan Victor Smith to be at the bedside giving comfort and confidence to the patient and his family in times of need. I had the privilege of being Alan's pathologist for a period of twenty years, and, by seeing many of his patients, I was able to judge the profound admiration, love and respect they had for him.

In these days when psychosomatic medicine is treated as a thing apart, it is refreshing to realize that many practitioners have an understanding of the patient as a whole and appreciate to the full that the prescription is not the end all of a doctor's task. Alan regarded each patient as a special and individual trust. He was extremely human, and his sympathetic approach, his eternal cheerfulness, his interest in the general well-being of the patient, his timelessness, his disinterest in himself, his skill and experience made him the perfect family doctor. His passing has left a deep effect on a large number of people in the eastern suburbs who feel that an integral part of their life has been taken away from them. Alan knew that the moving finger was soon to close his account, but he refused to spare himself and insisted on visiting his bedridden patients rather than restrict himself to staying in his rooms. The only honour which falls to men like Alan is the esteem in which he is held by all who knew him.

He was educated at Fort Street Boys' High School and won the honour cup in his last year at school. After graduating in medicine he joined Dr. John Meredith, who had the highest regard for him, firstly as an assistant and later as a partner at Weston on the South Maitland coalfields. In 1936 he joined Dr. Ken Addison in partnership at Bondi Junction, Sydney, and continued so till 1951 when Ken went to live at Lane Cove. A little later Dr. D. Hipsley joined him, and in 1954 Dr. J. Wells made a third partner. In 1953 Dr. Smith had a coronary from which he slowly recovered, and later several minor attacks before a suddenly fatal one.

He is survived by his wife and two daughters. Words are hollow things to express the deep feeling and respect one has for so exemplary a life.

Post-Graduate Work.

SYDNEY HOSPITAL SEMINARS.

Final Programme, 1958.

THE following seminars will be held on Wednesday, from 2 p.m. to 3 p.m., in the Maitland Lecture Hall at the Sydney Hospital. Each seminar will be preceded by medical grand rounds at noon (Maitland Lecture Hall), lunch in the Board Room at 1 p.m., and a pathological demonstration in the Maitland Lecture Hall at 1.30 p.m.

September 3, "Thyroiditis", Dr. R. Jeremy and Dr. J. M. Garven (St. Vincent's Hospital); September 10, "Artificial Kidney: Sydney Hospital Experience", Dr. H. M. Whyte and Dr. K. D. G. Edwards; September 17, "Recent Pharmacological Research in Sydney", Professor R. H. Thorp, University of Sydney; September 24, no seminar—Sydney Hospitallers' Week; October 1, "Stenosing Cholangitis", Professor F. A. R. Stammers, department of surgery, University of Birmingham, England (by courtesy of the Post-Graduate Committee in Medicine); October 8, "Atomic Medicine", Dr. G. Watson, head of Medical Section, Australian Atomic Energy Commission; October 15, "Present Status of Surgery in Broncho-Pulmonary Disease", Mr. W. G. Ferguson, pulmonary clinic; October 22, "Cerebral Vascular Disease", Dr. W. Wolfenden, neurology clinic; October 29, "Renal Tract Infection", Dr. J. N. Sevier and Mr. J. E. Blackman.

Your attendance and active interest in the above activities would be greatly appreciated.

THE MELBOURNE MEDICAL POST-GRADUATE COMMITTEE.

PROGRAMME FOR SEPTEMBER, 1958.

Visit of Professor F. A. R. Stammers.

PROFESSOR STAMMERS, C.B.E., T.D., B.Sc., F.R.C.S., of the Department of Surgery, Birmingham, is the category A lecturer for 1958 of the Australian Post-Graduate Federation in Medicine. He will visit Melbourne from August 30 to September 13 and carry out the following programme.

Metropolitan Lectures.

September 1, "Carcinoma of the Stomach"; September 4, "Pain in the Distribution of the Brachial Plexus"; September 9, "Peripheral Arterial Disease"; September 11, "Complications of Partial Gastrectomy: (*a*) Immediate, (*b*) Delayed".

The lecture on September 4 will be delivered at 5.15 p.m. in the R.C.O.G. Hall, 8 Latrobe Street. The other three will be at 8.15 p.m. in the B.M.A. Hall, 426 Albert Street, East Melbourne.

Hospital Visits.

September 2, Prince Henry's Hospital, 10 a.m. to 3.30 p.m.; September 4, Royal Melbourne Hospital, 10 a.m. to 3.30 p.m.; September 5, Royal Melbourne Hospital, 9.30 a.m., professorial ward round, and lecture at 1 p.m. on "Stenosing Cholangitis"; September 9, Repatriation General Hospital, Heidelberg, 10 a.m. to 3.30 p.m.; September 10, St. Vincent's Hospital, 10 a.m. to 3.30 p.m.; September 11, Alfred Hospital, 10 a.m. to 3.30 p.m.; September 12, Alfred Hospital, 10 a.m. to 3.30 p.m.

Country Lecture.

Professor Stammers will deliver a lecture at Hamilton on September 6 (see under "Country Courses").

Visit to the Royal Australasian College of Surgeons.

On September 12, at 5.15 p.m., Professor Stammers will lecture on "Researches in the Department of Surgery, University of Birmingham".

Country Courses.

Hamilton.

On Saturday, September 6, the following lectures will be given at the Glenelg Hospital:

3.45 p.m., "The Indications for and Results of Sympathectomy", Mr. R. S. Hooper; 5 p.m., "Some Experiences with Surgery of the Bile Duct, with Special Reference to Stenosing Cholangitis", Professor F. A. R. Stammers; 8.15 p.m., "Treatment of Psychosomatic Disorders", Dr. R. K. Doig.

Dr. R. R. Sobey, 6 Spence Street, Warrnambool, is the local secretary. Telephone 317.

Mildura.

On Saturday and Sunday, September 20 and 21, the following lectures will be given at the Base Hospital.

Saturday, September 20: 2 p.m., "Albuminuria", Dr. J. L. Frew; 4 p.m., "Colostomy", Professor Maurice Ewing. This lecture will be followed by a short talk by the professor on the researches in his department.

Sunday, September 21: 9.30 a.m., "Psychosomatic Gynæcology", Dr. Kelvin Churches; 11 a.m., "Lumps in the Breast", Mr. T. Ackland. This lecture will be followed by a quiz session.

Dr. T. K. Durbridge, 123 Langtree Avenue, Mildura, should be contacted regarding this course.

Swan Hill.

On Saturday, September 27, the following course will be held at the High School:

2 p.m., "Maternal Mortality", Professor Lance Townsend; 3.15 p.m., "The Coughing Child", Dr. H. N. B. Wettenhall; 4.45 p.m., "Intestinal Obstruction", Mr. H. H. Eddey.

Dr. W. Weaver, 16 Beveridge Street, Swan Hill, is the local secretary.

Flinders Naval Depot.

On September 10, at 2.30 p.m., Dr. J. Madigan will speak on "Radiation Therapy and Hazards". This meeting is being held by arrangement with the Royal Australian Navy.

Lecture in the Scientific Basis of Medicine.

All members of the profession are invited, without fee, to attend the lecture by Dr. A. G. Goble on "Aspects of Myocardial Ischæmia" at 8 p.m. in the Main Lecture Theatre, Royal Melbourne Hospital, on Friday, September 5.

Course in Psychiatry.

The course in psychiatry will close at the end of September. The following lectures on organic approaches in psychiatry will be given during the month at 8.15 p.m. in the Small Lecture Theatre, Royal Melbourne Hospital:

September 4, "E.E.G. and its Applications", Dr. A. Stoller; September 11, "Psychiatric Complications of Head Injury", Mr. R. S. Hooper; September 18, "The Chronic Insane Patient", Dr. H. M. Bower; September 25, "The Psychiatrist in Court", Mr. Justice Barry.

Recorded Lecture Library.

The following recorded lecture is now available on request from the Post-Graduate Committee for use with microgroove equipment. Country borrowers have priority.

"Metabolic Diseases of Bone", by Dr. M. D. Milne, of London. Two 10-inch disks, 17 slides (2" × 2").

INFORMATION.

The fees for attendance at the lectures to be given by Professor Stammers and at country lectures are at the rate of 15s., payable to the Post-Graduate Committee, but those who have paid an annual subscription to the Committee are invited without further charge. The address of the Melbourne Medical Post-Graduate Committee is 394 Albert Street, East Melbourne. Telephone: FB 2547.

The Royal Australasian College of Physicians.

VICTORIAN STATE COMMITTEE.

Lecture by Professor Ennor.

THE Victorian State Committee of The Royal Australasian College of Physicians has arranged for Professor A. H. Ennor, Professor of Biochemistry at the John Curtin School

of Medical Research, the Australian National University, Canberra, to deliver a lecture entitled "The Biochemistry of the Guanidines" in the lecture theatre of the Royal Australasian College of Surgeons, Spring Street, Melbourne, at 5.15 p.m. on Thursday, October 9, 1958.

All members of the medical profession are invited to this lecture.

Scientific Meeting.

The Victorian Fellows and members of The Royal Australasian College of Physicians will hold a scientific meeting at the Austin Hospital, Heidelberg, on Saturday, October 25, 1958. The programme will be as follows:

11.30 a.m., "Chemotherapy of Tuberculosis", Dr. P. R. Bull; 12 noon, "Pulmonary Function", Dr. Malcolm Allen; 12.45 p.m., luncheon; 1.45 p.m., "The Changing Concepts in the Diagnosis and Management of Pleural Empyema", Dr. John Clarebrough; 2.15 p.m., "The Bronchial Tree in Pulmonary Tuberculosis", Dr. C. H. Fitts; 2.45 p.m., "Sarcoidosis", Dr. T. H. Steel; 3.15 p.m., afternoon tea; 3.45 p.m., "Modern Trends in the Treatment of Bronchiectasis", Dr. Brian Marks; 4.15 p.m., "Demonstration of Equipment for Lung Function Studies and the Engstrom Respirator", Dr. Gordon Price.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes, etc., are published in the *Commonwealth of Australia Gazette*, No. 33, of June 12, 1958.

NAVAL FORCES OF THE COMMONWEALTH.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Volunteer Reserve.

Termination of Appointments.—The appointments of the following are terminated to date, 31st March, 1958: Surgeon Lieutenant-Commander Roland Frederick Kingston; Surgeon Lieutenant Rodney Ian Meyers.

AUSTRALIAN MILITARY FORCES.

Citizen Military Forces.

Northern Command.

Royal Australian Army Medical Corps (Medical).—1/55673 Captain (provisionally) G. F. Dixon relinquishes the provisional rank of Captain, 29th January, 1958, and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Northern Command) in the honorary rank of Captain, 30th January, 1958.

1/39085 Captain (provisionally) G. C. T. Kenny relinquishes the provisional rank of Captain, 2nd February, 1958, and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Northern Command) in the honorary rank of Captain, 3rd February, 1958.

1/61860 Captain (provisionally) W. A. Campbell relinquishes the provisional rank of Captain, 9th March, 1958, is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Northern Command), and is granted the honorary rank of Captain, 10th March, 1958. To be Captain (provisionally), 16th April, 1958: 1/39219 John Sylvester Harte.

Eastern Command.

Royal Australian Army Medical Corps (Medical).—The provisional rank of 2/146614 Captain K. F. Hume is confirmed. 2/146527 Lieutenant-Colonel J. F. C. C. Cobley is appointed from the Reserve of Officers, 1st January, 1958. 2/147990 Honorary Captain P. P. Manzie is appointed from the Reserve of Officers, and to be Captain (provisionally), 25th March, 1958. 2/127043 Captain (provisionally) A. R. Doutreband relinquishes the provisional rank of Captain, 23rd March, 1958, and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Eastern Command) in the honorary rank of Captain, 24th March, 1958. To be Captains (provisionally): 2/206961 Raymond Stanley Hyslop, 28th March, 1958, and 2/130123 John Henry Steele-Smith, 16th April, 1958.

Southern Command.

Royal Australian Army Medical Corps (Medical).—3/139406 Captain (provisionally) M. R. Barrett relinquishes the pro-

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED AUGUST 2, 1958.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	1	4(3)	13(1)	..	..	..	..	..	18
Amœbiasis	..	1(1)	..	..	1(1)	..	..	..	2
Ancylostomiasis	1	..	..	..	..	..	6	..	7
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	1	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	2(1)	23(22)	4(3)	..	..	..	..	2	31
Diphtheria	..	1(1)	1	..	2(2)	..	..	..	4
Dysentery (Bacillary)	..	3(3)	..	1(1)	2(2)	..	2	..	8
Encephalitis	..	..	..	..	..	..	..	1	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	1	..	..	1
Infective Hepatitis	63(29)	13(5)	6	4(2)	2	8(1)	4	..	100
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	1(1)	..	..	..	..	..	1
Leptospirosis	..	..	4	..	..	..	..	..	4
Malaria	..	..	1[2]	..	..	..	..	..	1[2]
Meningococcal Infection	1	2(1)	2	..	..	..	..	..	5
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	1(1)	..	1(1)	..	..	..	..	2
Paratyphoid	1(1)	..	..	..	..	..	..	..	1
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	..	..	..	..	..	..	..	..	..
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	17(11)	1	3	118(108)	..	..	33	172
Salmonella Infection	..	..	..	..	1(1)	..	..	..	1
Scarlet Fever	13(8)	22(20)	3(1)	..	3(2)	1	..	2	44
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	1	..	3	..	4
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	36(23)	23(17)	12(5)	5(5)	11(8)	1(1)	3	1	92
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

[2] Source of infection outside Australia.

visional rank of Captain, 31st December, 1957, is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Southern Command), and is granted the honorary rank of Captain, 1st January, 1958. 3/101826 Captain L. K. Morgan is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Southern Command), 1st March, 1958. To be Lieutenant-Colonel, 2nd January, 1958: 3/82441 Major (Temporary Lieutenant-Colonel) D. C. Cowling.

Central Command.

Royal Australian Army Medical Corps (Medical).—4/31903 Lieutenant-Colonel C. M. Gurner is appointed to command 1st Casualty Clearing Station, 1st February, 1958. 4/31911 Lieutenant-Colonel F. E. Welch relinquishes command 1st Casualty Clearing Station, 31st January, 1958, and is borne supernumerary to the authorized establishment of Lieutenant-Colonels with pay and allowances of Major (at own request), 1st February, 1958. 4/35233 Lieutenant-Colonel J. D. Rice is seconded whilst in the United Kingdom, 1st June, 1957. 4/31903 Lieutenant-Colonel C. M. Gurner ceases to be seconded whilst undergoing post-graduate studies in the United Kingdom, 31st May, 1957. 4/35233 Lieutenant-Colonel J. D. Rice ceases to be seconded whilst in the United Kingdom, 31st January, 1958. To be Lieutenant-Colonel, 30th January, 1958: 4/31911 Major (Temporary Lieutenant-Colonel) F. E. Welch.

Western Command.

Royal Australian Army Medical Corps (Medical).—5/26553 Captain (provisionally) P. D Goatcher relinquishes the provisional rank of Captain, 17th March, 1958, is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Western Command), and is granted the honorary rank of Captain, 18th March, 1958.

5/45806 Captain (provisionally) J. M. Lubich relinquishes the provisional rank of Captain, 29th March, 1958, is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Western Command), and is granted the honorary rank of Captain, 30th March, 1958.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps (Medical).

Southern Command.—The resignation of Honorary Captain A. W. Stapleton of his commission is accepted, 25th February, 1958. To be Honorary Captains: Ian Peter McIntyre and John Anthony Miramis, 10th February, 1958, and Peter Brayton Brown, 10th March, 1958, and Ian Irving Findlay, 11th March, 1958.

The following officer is placed upon the Retired List (Central Command) with permission to retain his rank and wear the prescribed uniform, 30th April, 1958: Major R. H. Hamilton.

The following officers are placed upon the Retired List (Northern Command), 30th June, 1958: Major A. K. Green and Captain W. J. Rawlings.

Notice.

THE COLLEGE OF PATHOLOGISTS OF AUSTRALIA.

Annual Meeting, 1958.

THE annual meeting of the College of Pathologists of Australia will be held at the Royal Society Hall, at the corner of Victoria and Exhibition Streets, Melbourne, on August 28, 29 and 30, 1958. Scientific sessions will take place on Thursday, August 28, from 9.30 a.m. to 4.45 p.m., on Friday, August 29, from 11 a.m. to 4.50 p.m., and on Saturday, August 30, from 9.30 a.m. to 10.30 a.m. The annual general meeting will be held on Saturday, August 30, at 11 a.m.

Nominations and Elections.

THE undermentioned have applied for election as members of the South Australian Branch of the British Medical Association:

Wyness, William John, M.B., Ch.B., 1935 (Univ. Aberdeen), D.M.R.D., Repatriation General Hospital, Springbank.

Russell, Frank Wylam, M.R.C.S., L.R.C.P. (London), 1951, 759 Port Road, Woodville.

Tang, Teng Kooi, M.B., B.S., 1958 (Univ. Adelaide), 9 Hexham Avenue, Myrtlebank.

Symonds, Edwin Malcolm, M.B., B.S., 1957 (Univ. Adelaide), 79 Kensington Road, Norwood.

The undermentioned have been elected as members of the South Australian Branch of the British Medical Association: Brown, Shelagh, M.B., Ch.B., 1950 (Univ. Sheffield); Urban, Marianne, M.B., B.S., 1958 (Univ. Adelaide); Neate, Robert James, M.B., B.S., 1957 (Univ. Adelaide).

Deaths.

THE following death has been announced:

HEGGATON.—Rupert Dufty Heggaton, on August 9, 1958, at Bellevue Hill, New South Wales.

Diary for the Month.

AUG. 26.—New South Wales Branch, B.M.A.: Hospitals Committee.
AUG. 27.—Victorian Branch, B.M.A.: Council Meeting.
AUG. 28.—New South Wales Branch, B.M.A.: Branch Meeting.
AUG. 28.—Queensland Branch, B.M.A.: Bancroft Oration.
AUG. 28.—South Australian Branch, B.M.A.: Scientific Meeting.
AUG. 29.—Queensland Branch, B.M.A.: Annual Fork Dinner.
AUG. 20.—Queensland Branch, B.M.A.: Annual General Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales. Anti-Tuberculosis Association of New South Wales. The Maitland Hospital.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

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